THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

JULY, 1937

COMMUNICATIONS

THE PROGNOSIS IN PAPILLOEDEMA*

BY

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LONDON

PAPILLOEDEMA is one of the common conditions which is a meeting ground of the ophthalmologist and the neurologist, and for academic as well as practical reasons concerns the practice of both, and no aspect of this condition demands more attention than its prognosis as regards the preservation of vision.

I am aware that in speaking to you to-day on the *Prognosis in Papilloedema* I am dealing with a subject which has been repeatedly discussed by this Society; in our *Transactions* you can find noteworthy contributions to it, particularly by Dr. James Taylor and Mr. Leslie Paton. Mr. Paton's articles on Papilloedema are in fact so comprehensive and based on such extensive experience that I fear I can add little to the conclusions he has already drawn. My excuse for introducing the subject again is that I frequently find that the presence of papilloedema is regarded as an immediate danger to vision. For instance, recently in an examination for a higher medical diploma I presented to several candidates a case showing early swelling of the discs, and was

* Presidential Address, Annual Congress, Ophthalmological Society of the United Kingdom, 1937.

told by almost all of them that steps should be at once taken in order to save vision. There is obviously, therefore, considerable misapprehension on the matter of prognosis among our colleagues in different branches of our profession, and it is on us the duty of instruction rests.

It is my intention to discuss to-day only that form of papilloedema associated with increased intracranial pressure and to exclude the question of its diagnosis. Its pathology need not delay us, for it is now recognised by everyone that it is essentially an oedema of the nerve head in which, for a time at least, there is little structural damage or functional disturbance of the nervous elements, though these may be strangled and destroyed by overgrowth of interstitial tissue when the oedema subsides into atrophy. Unfortunately we must also recognise that in this stage no recovery of vision can be expected.

As the essential aetiological factor in papilloedema is increase in intracranial pressure, the relief of this pressure will lead to its disappearance and remove the risk of blindness or serious deterioration of vision if it can be effected before secondary changes have developed in the disc. It is therefore natural that those who are primarily concerned in the conservation of the patient's sight should demand early or immediate operation, for it is only by operation that permanent relief of pressure can be obtained. But there are often cogent reasons against immediate operation. Complete removal of the tumour, which is the condition with which papilloedema is most often associated, is frequently impossible, more especially as it is in cases with extensive infiltrating tumours of the glioma type that the severer degrees of papilloedema are found, and in these cases operation frequently reduces the length and utility of life. Even a cerebral decompression often leads to considerable physical disablement. Further, in even the most experienced hands operation, whether radical or palliative, entails considerable risk to life, and for this reason or on other grounds, as the lack of symptoms that can establish a definite localisation, delay is often advisable.

We are consequently often faced with the questions: What will happen if pressure is not relieved at once? and, How long can we delay without endangering the patient's vision? To these questions no general answer can be given; each case must be considered on its own merits. But the statement in one of the most recent textbooks on neurology that " every longstanding case of papilloedema inevitably leads to blindness " cannot be accepted without qualification.

It is of course known to every ophthalmologist that even intense papilloedema may not cause any disturbance of vision; Hughlings Jackson recognised this a few years after the introduction

of the ophthalmoscope and emphasised it again in his Presidential address to this Society 48 years ago. It is not, however, so generally recognised that vision may remain unimpaired for long periods, that in other words the papilloedema may be non-progressive. It is a frequent experience of neurologists that even in the presence of considerable congestion and swelling of the optic discs there may be no deterioration of sight for months, and occasionally we meet with cases in which vision remains unchanged for years. In the earlier volumes of our *Transactions* such cases have been described by Hartridge, Marcus Gunn, Frost and others, and Gowers, an unrivalled observer, stated in his *Medical Ophthalmoscopy*, " The time taken for the development of changes varies within wide limits. A neuritis may remain for months or even for years unchanged, or the most intense strangulation may be developed in a few weeks," and on another page he wrote " at any stage the morbid process may stop, remain stationary for a long time and then recede."

I have seen many such cases in which the existence of a papilloedema was beyond doubt and yet vision remain unimpaired for long periods. One man, for instance, whom I was able to follow for six or seven years, preserved normal visual acuity and unrestricted visual fields. It is true such cases are exceptional, but it should be recognised that they do occur. In these cases there are probably either quiescent or slowly growing tumours to the presence of which the intracranial contents can adapt itself so that there is no great or sudden rise of pressure.

In other, but rarer, cases the papilloedema subsides without surgical intervention or other specific treatment, and vision remains unaffected. Hughlings Jackson described several such cases and Gowers wrote many years ago, " now and then a neuritis rapidly subsides although the cerebral disease progresses." In a later volume of our *Transactions* Mr. Maitland Ramsay referred to " a considerable number of cases in which, in spite of intense oedema, the condition did subside, vision was unimpaired and no operation became necessary." I have personally followed several such cases. In most of them it is true the swelling of the discs was low, and hæmorrhages and exudates were absent or scanty, but this is not always so. One of the most striking instances was a doctor's wife in whom several expert ophthalmologists had no doubt that papilloedema with considerable swelling and hæmorrhages existed. Its association with headache suggested the presence of an intracranial tumour, though she showed no other signs of cerebral disease. Though no operation was undertaken the papilloedema subsided slowly, vision remained intact and there has been no recurrence or the development of other symptoms during the past four years.

In most of these cases it is impossible to ascertain the exact nature of the condition to which the papilloedema is due; in some there may be a tuberculous or gummatous tumour which becomes quiescent, or a gliomatous growth which undergoes degeneration, but internal hydrocephalus is probably responsible in many of them for the elevation of intracranial pressure, and in others there may be an external hydrocephalus or "serous meningitis," that is an excess of cerebrospinal fluid in the sulci and on the surface of the brain, such as is frequently found in association with otitis and other infective processes in the skull.

It is true that spontaneous subsidence of papilloedema is rare and that non-progressive papilloedema is not common, but their occurrence and the fact that swelling and congestion of the discs may persist for long periods without serious deterioration of vision, shows that papilloedema as such does not always demand immediate surgical intervention.

In most cases of papilloedema, however, the relief of intracranial pressure is, or soon becomes, necessary to save sight, and we are consequently faced with the important question: what indications are there?, or what warning may we have, that further delay is dangerous? In the first place there can be no doubt that the more rapidly the congestion and swelling of the optic discs develop, the more intense does the papilloedema become and the greater is the danger to sight if the intracranial pressure is not relieved. Swelling of five or more dioptres is usually a danger-signal against undue delay. Intense congestion, as shown by great engorgement of the retinal veins and early and extensive hæmorrhages on the surface of the swollen disc, are equally serious features, in fact they may be regarded as the most important measure of the severity of the process. The early appearance of exudates in the form of large soft greyish patches on the swollen discs, or of a macular fan, are also significant signs, though they may not themselves interfere with vision. An even more important danger-signal is, as Mr. Paton and others have pointed out, a narrowing or decrease in calibre of the arteries in the swollen disc and its neighbourhood, in fact when the narrowing is pronounced it is improbable that vision will remain unimpaired even though cerebral decompression is at once undertaken. Contraction of the arteries may be in fact regarded as the first evidence of constriction of the structures in the nerve-head by the organising fibrous and glial tissue, and irreparable damage of the nerve fibres soon follows. The appearance of white lines along the vessels usually belongs to a later stage of consecutive atrophy.

In the later stages increasing greyness and density of the swollen disc, a subsidence of the swelling accompanied by even the slightest deterioration of visual acuity, or commencing con-

striction of the visual fields, are indications that in the near future vision may be affected by the histological changes associated with consecutive atrophy. Of these signs of an oncoming atrophy special importance must be given to peripheral contraction of the visual fields; it is often the most prominent of the symptoms associated with early atrophy and frequently the first indication of secondary changes.

To you it is unnecessary to emphasise the importance of frequent and careful observations to follow the evolution of the fundal changes and to detect the earliest signs of secondary atrophy which constitute such a serious threat to vision.

Another symptom which is generally regarded as a warning that vision is in danger is the transient loss of sight of which many patients complain. It may be only momentary, but occasionally it *lasts* for a few minutes, or on rare occasions it may pass off even more slowly. It has been attributed by some to a temporary increase of congestion, and by others to a temporary failure of circulation in the retinae and optic nerves, and the fact that it is often brought on by a sudden change in posture, as rising quickly from the prone or sitting position, favours the latter view. I agree, however, with Mr. Paton that it more probably results from compression of the chiasma and optic tracts by the bulging or distended floor of the third ventricle. It certainly occurs more commonly with tumours in the posterior fossa of the skull, which almost invariably lead to internal hydrocephalus, than with growths in other regions. The fact that it occasionally precedes the appearance of pathological changes in the discs or apart from them, as Dr. Hughlings Jackson and Mr. Paton have observed, an observation that I have been able to confirm, indicates that it is not directly due to papilloedema though often associated with it. When, however, this temporary amaurosis occurs in a patient with highly swollen discs it must be regarded as a threat of permanent failure of vision, as the intracranial as well as the intra-ocular changes with which it occurs, are usually so intense that sight may be soon endangered.

The treatment of papilloedema is of course removal of its cause, which is increased intracranial pressure, either by extirpation of the tumour or other pathological condition which raises pressure, or when this is not practicable, by a decompression craniectomy. As a rule the swelling and congestion of the discs subside within two to four weeks after operation, and when secondary changes had not appeared by then there is no further risk of impairment of vision. When visual acuity is seriously reduced before operation it is unfortunately rare for appreciable recovery to take place. When, however, the impairment of acuity is slight vision may improve considerably; I may recall to you that Mr. Paton observed

such improvement in one-third of a series of cases which he followed after operation, but my own experience has not been so fortunate. Visual fields that have become seriously constricted rarely recover fully, though a moderate degree of peripheral narrowing often disappears as the papilloedema subsides.

Occasionally there is immediately after operation a rapid deterioration of sight, which is, however, often transient; it is probably due to a sudden fall of pressure in the central artery of the retina as a result of too rapid relief of intracranial tension, and is therefore comparable to the diminution of vision which sometimes follows a successful operation for glaucoma.

Unfortunately vision may also fail permanently after a decompression operation in patients in whom neither the ophthalmoscopic changes nor the state of the visual fields had suggested the risk of such a catastrophe. This, in my experience, has been most common after either partial or complete removal of a tumour from the posterior fossa of the skull. For instance, I recently saw a woman with good visual acuity and unrestricted visual fields and a degree of papilloedema which could not be regarded as severe before operation, who, a few days after the partial removal of an acoustic nerve tumour and an extensive decompression, complained that her sight was failing and within a week or two was permanently blind. An equally tragic case was under my care recently. Visual acuity was 6/9 in each eye and the visual fields were full, and though the swelling of each disc amounted to about 5 dioptres the ophthalmoscope did not reveal any changes suggestive of impending atrophy. A fortnight after complete removal of a large dural endothelioma which compressed his left frontal lobe he complained of impairment of vision which progressed to complete blindness of one eye and left 6/36 vision only in the other. The swelling of the discs subsided slowly into a characteristic secondary atrophy.

Such progressive failure of vision after operation in cases in which it had not been expected or foretold is naturally distressing to all concerned, particularly when the cerebral operation promises permanent relief, but fortunately it is not common. The factors which determine the rapid secondary atrophy to which it is due are uncertain, and I fear I cannot suggest any signs which may warn us of its possible occurrence.

But an accurate prognosis of the state of vision after relief of intracranial pressure can usually be given by careful and if necessary repeated observations, though, as much else in medicine, a reliable judgment depends largely on experience, and as a guide to it we are often unable to formulate definite rules.

TARSORRHAPHIA MEDIALIS VERA

BY

N. I. SHIMKIN, M.D., PH.D.

HAIFA, PALESTINE

A NEW method of surgical treatment of long standing cases of ectropion paralyticum in lower eyelids is described below.

The author has applied this method of operation in a patient suffering from nerve leprosy. Ectropion paralyticum in the lower lid of the right eye developed in this patient about 17 years ago, and that of the left eye, about 19 years ago.

Groenouw[4] has pointed out that in this form of leprosy lagophthalmos paralyticus is observed in more than half the number of all cases, and appears during the first years of the disease; as nerve leprosy, like the present case, continues for years, lagophthalmos paralyticus gradually changes into severe forms of ectropion paralyticum, the consequence of which is keratitis e lagophthalmo, gradually leading to blindness owing to ulceration and drying up of the cornea.

Figures representing such leprous patients with different degrees of ectropion paralyticum may be found in the works of Groenouw (p. 1005), Manson[7] (p. 564), Elliot[2] (p. 300), Willy[9] (p. 568, Vol. II) and others. With respect to its severity, the present case resembles that of Groenouw.

At present, there are few lepers in Palestine, therefore occasions for treating a long-standing ectropion paralyticum leprosum are very rare. One of these cases was operated upon by me. Dr. Canaan[1], for many years director of Lepra Hospital in Jerusalem, pointed out in an article in 1927, that there were in Palestine about 80 patients with leprosy in different stages. In a private letter, dated November 22, 1936, he kindly informed me that in Palestine and Trans-Jordan, there were about 80-100 lepers; " The nodular and mixed forms are prevalent over the nervous form."

But, according to Rogers[8], in all the world there are from 2-3 million lepers, several hundred thousand of whom, probably, suffer, from consequences of lagophthalmos and ectropion paralyticum. Groenouw, Elliot and King indicate that in such cases the operation, tarsorrhaphia medialis, should be applied, the more so that " lepers tolerate operation upon the eyes very well," (King)[5].

However, long-standing ectropion paralyticum of the lower lids is met with not only in lepers. Paralysis facialis superior, due to quite different causes, may, in some cases lead to severe forms of long standing ectropion paralyticum of the lower lids, if a timely operation is not performed.

Loehlein[6], who had collected all the methods for surgical treatment of ectropion paralyticum of the lower lids by means of tarsorrhaphia medialis, points out, justly, that all these methods are applicable only in slight cases of lagophthalmos paralyticus, and that all the methods of tarsorrhaphia medialis offered, are actually only blepharorrhaphia medialis, as with all these methods, the tarsus of the lids is not included in the operation at all, and only a strip of skin round the inner canthus of the upper and lower lids of the eye is excised, then the skin edges are sutured together. In this way, the inner part of the paralysed lower lid is lifted and comes into closer contact with the eyeball, and, principally, the puncta and canaliculi of the lower lid are kept in position.

Fuchs[3] points out that in long-standing cases of ectropion paralyticum not only is the lower lid not kept in contact with the globe, but is everted outside in the inner canthus too; besides, it is elongated owing to its having stretched downwards.

In those long-standing cases, Fuchs simultaneously applies two operations upon the same lower lid; in the inner canthus, he performs blepharorrhaphia according to Arlt; and in order to shorten the lid, he excises a triangle on the outer third; then he sutures the sides of the triangle together.

The purpose of this double operation is to shorten the lower lid and keep the lacrymal passages intact.

But in long standing ectropion paralyticum as in our case, the punctum lacrimale is occluded to such an extent, that it is difficult to trace it even with a loupe; and even a thinnest conical probe cannot be inserted into the canaliculus lacrimalis. No restoration of lacrymal ducts can be thought of in these cases at all.

In those cases of ectropion paralyticum, where the lacrymal passages of the lower lid cannot be restored, the surgeon has only one aim, *i.e.*, to give the lower lid its normal position, in order to keep the cornea from drying up and ulcerating by covering it with the lid. Retention of tears in the conjunctival sac, resulting from the difficulty of their flowing away, in the above cases of ectropion, is rather a beneficial factor. The constantly wet cornea becomes more transparent and the vision improves.

In the case described below, I have noticed that, thanks to continuous moistening of the cornea by tears, the vision improved, the right eye beginning to distinguish the number of fingers at a distance of 4 metres, as against 1 metre before the operation; and the left eye, at that of $2\frac{1}{2}$ metres as against $\frac{1}{2}$ metre.

I beg to offer my method of operation, the tarsorrhaphia medialis vera, only for such long-standing cases of ectropion paralyticum.

This method differs from the tarsorrhaphia used up till now in that by its means the severest ectropion paralyticum medialis may

be corrected without any additional operation on the outer third of the lid as in the method of Fuchs.

This method is a true tarsorrhaphia, as in it, the tarsi of both lids in the inner part are sutured, and not the skin only as in the method of tarsorrhaphia suggested up till now. In this method, it is possible to lift the lower lid as high as the surgeon considers desirable.

The close fitting of the lid to the globe is well attained. Finally, the tarsi sutured together, provide a denser support to the lower lid and protect the eye more securely from a possible relapse of lagophthalmos. Besides this, the lacrymal passages of the upper lid remain intact.

The substance of the operation consists in the following: the conjunctiva having been freshened, the medial ectropionised part of the lower lid, freed from the skin, is drawn by means of two loop-like sutures inside a deep pocket, made by splitting the medial part of the upper lid into two plates.

The depth of the pocket in the inner part of the lid and the degree of drawing in of the lower lid inside it, are regulated by the surgeon during the operation.

Before describing all the steps of this simple operation in detail, I beg to cite the history of the disease in my case.

History.—October 25, 1936. The patient A.K., is 50 years old, a farmer (fellah). He does not remember the beginning of the disease. Red spots appeared on the forearm and on the wrists of both hands before the War (25-30 years ago). Soon similar spots appeared on shins and the soles of both feet. Then wounds began to appear on fingers and toes. The patient does not remember precisely, how long the wounds on hands and feet have continued, but only points out that first the fingers began to fall painlessly off, and then the toes. On the back of the left sole there is still a deep wound which has not been healing for 2 years.

The epiphora of the left eye began about 19 years ago, that of the right, 17 years ago. Soon the lower lids of both eyes began to roll out, first that of the left eye, and then of the right. At first there were many tears, and then the quantity of tears went on diminishing; the eye began "to dry up and darken." The vision has been decreasing. He distinguishes the road with difficulty. The patient came to the clinics with a request to keep his vision. He has been married 15 years. His wife is 30 years old, his son is 8. All the family are well.

State.—October 25, 1936. The patient is of medium height, has a very weak constitution. He represents a typical picture of nerve leprosy which in the present case differs in nothing from the typical cases described in the manuals mentioned above. As to the change in the region of the eye a complete degenerative atrophy of

all mimic muscles, draws our attention, together with an inveterate bilateral peripheral affection of the upper nervi faciales. Only the musculus supercilii dextri, as it is seen in the figure (photo 1) may be still voluntarily lifted up in its outer part, thanks to which the outer part of the right eyebrow is somewhat raised upwards. The I and II branches of the fifth nerve are also affected, owing to which a decreased thermal, tactile and pain sensibility of the skin, the face and also of conjunctiva and cornea, and a retarded winking are observed. On the part of the oculomotor nerve only a complete paralysis of the sphincter pupillae is observed, owing to which the pupils are greatly dilated and absolutely immobile. The upper lids, owing to the atrophy of the orbicularis palpebrarum, hang mechanically down, due to their weight, giving a picture of hemiptosis.

The lower lids are not in contact with the eyeball, and in their inner part hang down and are rolled out to such an extent that the puncta lacrimalia are lying as if on the cheek; the conjunctiva near them has a skin-like appearance. The puncta lacrimalia and canaliculi lacrimales are so occluded that it is impossible to insert into them the finest conical probe even with the help of a loupe. When the upper lids are lowered, the cornea in the lower third remains uncovered. The distance between the puncta of the upper lowered and lower lid in the right eye is 9 mm., and in the left eye 11 mm. The conjunctiva bulbi in the region not covered by the lower lid is dry and has a skin-like appearance. The cornea in the lower and middle thirds has grown opaque to such a degree that the pupil and iris are indistinctly seen. The upper third of the cornea is very slightly opaque and it is possible to see only the dilated and absolutely immobile pupil. There are no inflammatory processes in the anterior part of the eyeball. Owing to the opacification of the cornea no ophthalmoscopy is possible. All the movements of the eyeball are normal. Vision: R.E. = counting fingers at one metre distance. Vision: L.E. = counting fingers at a distance of $\frac{1}{2}$ metre. Tension—normal.

We will dispense with the analysis of separate symptoms of the above history of the disease, in order not to go beyond the boundaries of the title of the article; it should be only pointed out that in the present case there was a long-standing ectropion paralyticum leprosum which had reached its extremest degrees in the inner part of the lower lids of both eyes, which was aggravated by a total occlusion of puncta lacrimalia and canaliculi lacrimales. In consequence of a long-standing ectropion, drying up and opacification of the cornea had appeared, which led to a great decrease of the vision. The retarded winking and a diminution of the sensibility of the cornea and conjunctiva could cause a further opacification of the cornea and bring about a total blindness. Thus there were

present all the indications for an operation to correct the ectropion paralyticum of the lower lid.

When choosing the method of operation, it was necessary to take into consideration the presence in this case of the atrophy of the lids, muscles and tarsi, occurring in lepers (Groenouw), and besides, the degree of ectropion and principally the impossibility of restoring the lacrymal passages of the lower lid. All the operations blepharorrhaphia medialis, mentioned above, are applicable in slight cases of lagophthalmos; and in the present case could not give the required improvement of such a severe ectropion. The Arlt-Fuchs operation wounds the lid at its inner and outer thirds and atrophy of the tarsi and lids being present, this operation could not achieve a satisfactory result. Therefore, it was necessary to select an operative method which would effect the highest raising and shortening of the lower lid; its close contact to the globe, and the least trauma of the tissues of the lid.

The method of operation described below, tarsorrhaphia medialis vera, gives a complete correction of a long-standing ectropion, with the least trauma of the lid.

Description of the Operation

Instruments.—Fixation forceps, half-bent scissors, keratome, Jaeger's spatula, a sharp scalpel, needle-holder. 3 threads armed with needles at both ends, and 1 thread with 1 needle.

Preparation for the operation.—The skin of the lid and the inner part of the eye are washed well with soap and smeared over with iodine.

Anaesthesia.—2 c.c. sol. novocaine 2 per cent. + adrenalin are injected subcutaneously in the inner part of the lower and upper lids. 2 to 3 drops of sol. pantocaine 1 per cent., or sol. cocaine hydrochlor. 3 per cent. are instilled into the conjunctival sac.

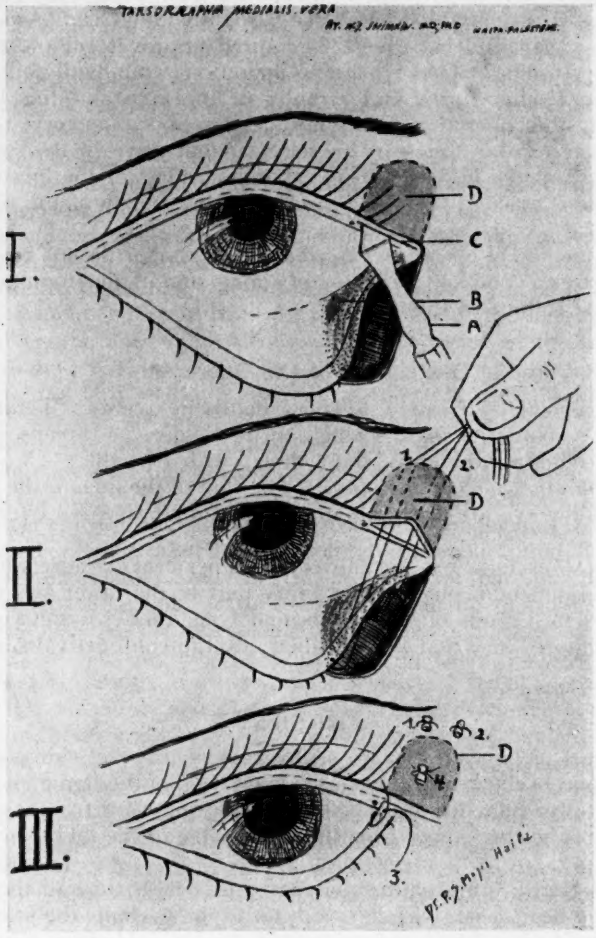
The Operation.—

1st Step

(Fig. I. A). Cutting off with scissors of the skin of the inner part of the lower lid from the beginning of the ligament down to the lower punctum lacrimale and 2 mm. outward from it. The width of the skin cut off from the actual edge of the lid downwards is $1\frac{1}{2}$ cm.

(B) Scarification of the margin of the lower lid and the conjunctiva near it along all its width up to the eyeball; the scarification begins from the inner canthus of the eye, including caruncula lacrimalis and plica semilunaris, down to the punctum lacrimale and 2 mm. outward from it.

(C) Splitting of the upper lid, along the intermarginal line into two plates, by means of the keratome, beginning from the punctum lacrimale superior to the inner canthus of the eye. When splitting, the punctum and canaliculus of the upper lid are kept intact. (For convenience of splitting the Jaeger hornplate may be laid under the border of the upper lid.)



FIGS. I, II, III.

(D) The slit produced is enlarged to 1½ cm. in height and width by means of the keratome or scalpel, thanks to which a deep pocket is produced in the inner part of the upper lid.

2nd Step

Table (Fig. II). *Application of sutures.* The needle of the first suture is introduced into the inner part of the lower lid 1 to 1½ mm. externally from the punctum and 1 mm. lower than the lid border; next to the first suture, 2 mm. medially, the second needle of the same suture is put through. The needles pierce the whole thickness of the lid from the outside inwards. The threads led out are introduced in the hollow of the pocket in the same order and piercing its top, go out over it. The distance between the threads carried outside is 2 mm. (Fig. II, 1).

The second suture (Fig. II, 2) is inserted in the same order in the lower lid, 3 mm. medially from the first suture; and both threads of the second suture are also introduced inside the pocket and go out over its top, 3 mm. medially from the first suture (Fig. II, 2). The threads having been brought out, all four are taken hold of by the thumb and forefinger and are drawn upwards. The inner ectropionised end of the lower lid is drawn into the hollow of the pocket, while the upper wall of the pocket is being raised upwards with forceps, thus enlarging the entry into it.

The surgeon uses his discretion whether to draw the threads tighter in order to raise the lower lid higher, or to slacken the threads, if the lid is shortened too much, and its contact with the eyeball is therefore too close. The surgeon being satisfied that the position of the lid is correct, the sutures are tied over a cotton-wool or gauze tampon (Fig. III, 1, 2). The result of the operation is seen immediately on tying the first and second sutures.

3rd Step

The newly formed inner canthus consists of 3 layers: 2 layers of the split upper lid and a layer of the tarsus and tissue of the inner part of the lower lid, drawn in between the first two. All the 3 layers are fixed together by means of a single suture in the following way:—

The needle pierces in the canthus all the three layers from inside out. This suture is tied over a gauze or cotton-wool tampon with a surgical knot. (Fig. III, 3.) In the figure the threads are free, the knot is not tied. Then to compress all the 3 layers to each other, a single loop suture is applied to the middle of the former pocket, in such a way that each needle of this suture is introduced under the newly formed inner canthus from inside out at a distance

of 2 mm. from each other. The threads of this suture are tightly tied over a gauze or cotton-wool tampon. (Fig. III, 4.)

The operation being over, 5 per cent. xeroform ointment and a pressure bandage are laid on both eyes.

Post Operation Period.—The first bandage is changed in 2 days. On the fifth day the sutures are taken out and a bandage is put only on the operated eye for two days more. After 7 days the patient

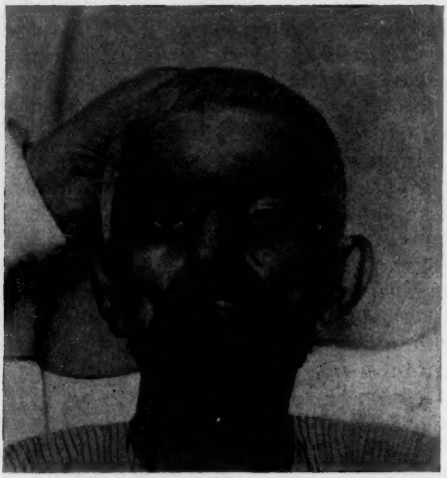


PHOTO 1.

is sent home. The result of the operation is seen both in Fig. III and in photo No. 1.

On the 8th day after the operation, *i.e.*, on November 2, 1936, the patient was demonstrated by me in the Society of Jewish Physicians in Haifa. On the next day after my report, Dr. Izkovich, the President of the Society and Director of the Roentgen Institute, kindly photographed the patient. (Photo 1.)

In the photo annexed, it is possible to see the close contact between the lower lids and the eyeball and a quite satisfactory cosmetic effect. The closing of the eyelids is nearly complete, in spite of the atrophy of the orbicularis of the upper lids usual in patients affected with nerve leprosy; such atrophy impeding the tight closing of the lids.

The therapeutic result of the operation was that the cornea of both eyes and the conjunctiva of the lower part of the eyeball were

copiously moistened with tears retained by the lower lid. It may be seen in the photo how the tears have gathered in the inner canthus of the right eye. The patient has noticed the disappearance of the feeling of dryness and burning in the eye and an improvement of vision too. The vision of the right eye on the 8th day after the operation was equal to counting fingers at a 4 metre distance (1 metre before the operation); that of the left eye 2½ metres (before the operation ½ metre). For the uncomplicated needs of a farmer (fellah) such vision is sufficient: it gives the patient the ability to move independently, without anybody's help.

The simplicity of the operation and attainment of a good covering for the cornea by means of raising and shortening the lower lid in the inner part of the eye, induced me to offer to the attention of ophthalmologists this new method, tarsorrhaphia medialis vera, in such long-standing cases of ectropion paralyticum where the lacrimal passages of the lower lid are so occluded that their physiological functions cannot be restored.

Summary

The author offers a new method of operation, tarsorrhaphia medialis vera for cases of ectropion paralyticum of the lower lid where the punctum and canaliculus lacrimalis are so occluded that the physiological function of the lower lacrymal passage cannot be restored.

The operation consists in drawing the inner part of the lower lid, from the punctum lacrimale up to the inner canthus, by means of two loop sutures upwards and inside a deep pocket, made in the inner part of the upper lid; the pocket is made by means of splitting the margin of the upper lid along the inter-marginal line into two plates, beginning from the punctum superior and up to ligamentum internum. The punctum and canaliculus of the upper lid remain intact in this operation.

On the medial part of the lower lid the skin is previously excised, and the conjunctiva and lid margin are scarified, which causes its adhesion, when healing, to the tissues of the upper lid pocket walls. All the three layers are fastened together with a single knot suture in the newly formed canthus of the eye, and with a loop stitch inwards from it. (Fig. III, 3, 4.)

The operation is called tarsorrhaphia medialis vera, as according to its method the tarsi of both lids are sutured in the inner eye canthus, and not only the skin of the lids, as occurs in all the methods of tarsorrhaphia medialis, devised up till now. This operation gives the surgeon the possibility of gauging the amount to raise and shorten the lower lid according to what is necessary. The cosmetic result is quite satisfactory.

REFERENCES

1. **Canaan, T.**—Die Lepra in Palaestina. *Archiv fur Schiffs- und Tropenhygiene*, S. 21, H. 1, Bd. XXXI, 1927.
2. **Elliot, R. H.**—Ophthalmologie Tropicale. *Traduction Française*, 1922.
3. **Fuchs, E**—*Lehrb. d. Augenheilk.*, S. 997, XIII Aufl., 1921.
4. **Groenouw, A.**—Beziehungen der Allgemeinleiden und Organerkrankungen zu Veranderungen und Krankheiten des Sehorgans. Abteilung 1. *Graefe-Saemisch Handb. d. Augenheilk.*, III Aufl., 1920.
5. **King, E.**—The Eye in Leprosy. *Brit. Jl. of Ophthal.*, Oct., 1936, p. 568.
6. **Loehlein, W.**—Operationen an den Augenlidern. *Graefe-Saemisch Handb. d. Augenheilk.* Augenaerztliche Operationslehre I, S. 162. Zweite und dritte Auflage, 1922.
7. **Manson, S.**—Tropical Diseases. X Edition, 1935.
8. **Rogers, L.**—Leprosy. *Encyclopaedia Britannica*, Vol. XIII, p. 957, 1929.
9. **Willy, Alexander.**—Spezielle Pathologie und Therapie innerer Krankheiten, herausgegeben von Friedrich Kraus und Theodor Brugsch. X Bd., 1 Teil.

THE WATER-BINDING OF THE BRAIN

BY

J. A. VAN HEUVEN *and* P. F. FISCHER

UTRECHT

IN our last paper on " The Water-Binding of the Optic Nerve and of its Sheaths," we came to the conclusion that the optic nerve is capable of taking and keeping such quantities of water that this capability may play some part in the origin of a papilloedema. As the result of their pathologico-anatomical investigations in this field, Spatz and Marchesani came to the conclusion that papilloedema can be compared with the " Zysternenverquellung " which was seen by Spatz in the brains of patients suffering from brain tumour. If this conclusion is accurate it follows necessarily that the water-binding power of the brain tissue must be similar to that of optic nerve. Now it is a matter of common knowledge that brain tissue absorbs water, and the expression " oedema of the brain " is commonly used. In both this question, however, and in the use of the term " papilloedema " one is inclined to think solely of the histological picture and not to consider the physico-chemical conditions.

Some investigations were made formerly by M. H. Fischer and his collaborators in the absorption of water by nervous tissues and brain tissues of rabbits. These authors worked with complete sets of brains, and their investigations were limited to a small number of experiments. They arrived at the important conclusion that brain tissues are quite capable of absorbing water, especially in weak acid solutions, and furthermore that an increase of weight of only 10 per cent. of brain tissue was sufficient to

cause a fatal brain oedema. In order to obtain a clearer insight into the rôle played by the physico-chemical properties of the brain tissue in the pathogenesis of the papilloedema, we have performed the experiments which follow.

It speaks for itself, that brain substance as a whole shows a different behaviour from separate sections of the brain. In the same way the quantity of water which can be absorbed by a whole eye is quite different from that which can be absorbed by isolated parts of the eye. Investigations were made separately of the water-binding of the grey and of the white brain matter. It was impossible to say beforehand whether, *e.g.*, the white brain tissue would react similarly in and from different anatomical situations in the same brain. We therefore aimed to take always the tissue from similar regions of the brain, doing so both in white and grey matter. Our white tissue was taken from the tissue between the internal capsule and cortex and our grey matter from the basal ganglia.

The second important fact was that for our investigations we had to limit ourselves to pieces as small as possible as to surface, weight and volume. We endeavoured to take pieces of brain as similar as possible in size to those from the optic nerve referred to in our paper on that subject (*Brit. Jl. of Opthal.*, April, 1936). As we explained in the above-mentioned paper, it is important always to take pieces of tissue of the same size, as only by so doing are comparisons made possible. In order to make this paper a continuation of that on the optic nerve we acted as follows:—

Again we used ox brains, obtained as fresh as possible from the slaughter house and put these brain masses on their bases, cutting them into halves, by means of a large knife, in such a way that the basal ganglia were divided into equal parts. We took always first the white brain matter and after that the grey; in both cases small pieces of 2 mm. thickness and 4 mm. length. By doing so we got pieces of similar size and approximately similar weight to those taken from the optic nerve in our previous paper. These pieces were laid in little metal baskets, which were standing on a tripod in a weighing glass of known weight. As a matter of fact the same glasses were used, which we also used in our former experiments on the water-binding of the retina and of the optic nerve, which are described in our paper on the water-binding of the retina in the July, 1935, issue of this journal.

We took special care that the brains obtained were as fresh as possible, as we realised very soon that brain substances which are old or even brain substances which have been in the refrigerator for some time absorbed decidedly less water. This fact must be explained by the high quantity of acid the brain

contains, making brain substance very much inclined to absorb water from its surroundings. Thus if the brain matter is not quite fresh, a considerable water-absorption will have already taken place. For the same reason brain substance which is not quite fresh is heavier than fresh.

As was done in our investigations into the water-binding of the retina and the optic nerve, we started our investigations on the water-binding of the brain by studying the water-absorption from pure water. Figure 1 shows our results. The ordinates show the percentage increase in weight through water absorption and the abscissae the time in hours. The diagram shows the conduct of the white and grey brain substances in distilled water and at the same that of the optic nerve, of the retina and of the sheaths of the optic nerve. Thus the difference in behaviour of the different tissues is shown clearly. It appears that the curve, which shows the water-absorption out of pure distilled water by the grey brain substance, is very similar to that of the optic nerve. The optic nerve takes a higher percentage of water than the grey matter, and a much lower percentage than the white matter. The water-binding of the sheath is absolutely and relatively much greater than that of all other tissues, and conversely the water-binding of the retina is both absolutely and relatively the smallest.

Observing the behaviour of the different tissues within the first few hours, we find the following series:—

Sheath of the optic nerve larger than optic nerve. Optic nerve larger than white brain matter. White brain matter larger than grey brain matter. Grey brain matter larger than retina.

In this series the tissues with the greatest water-binding power are given on the left hand and those with the smallest water-binding power on the right. The percentage of difference is greatest between retina and sheath of the optic nerve and is as much as 64 per cent. This figure represents between optic nerve and retina 39 per cent., between optic nerve and grey brain matter 23 per cent. and between white and grey brain matter 8 per cent.

Based upon these investigations we conducted further experiments tracing the conduct of the brain in acid. Again we used the same acids in the same concentrations as we used previously, in dealing with retina and optic nerve. The only difference in comparison with the optic nerve is, that in the next diagram the figures mentioned are not those found after 3 hours but those found after 24 hours. This could easily be done as in dealing with brains we had much more material at our disposal than in dealing with previous tissues. This means that we have done our tests after one, three, six and 24 hours respectively, but the diagram

shows that the figures found after 24 hours were in logical agreement with those after one, three and six hours. Seeing we obtained such a regular curve as is shown in Figure 1, we kept all our small sections of tissue in the different bathing liquids for 24 hours after our first preliminary experiment. In this way we were able to check our totals after 24 hours, and this explains why we show these figures.

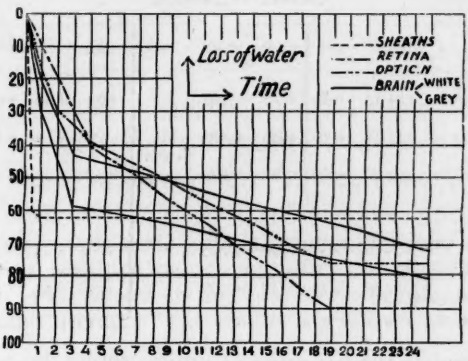


DIAGRAM I.

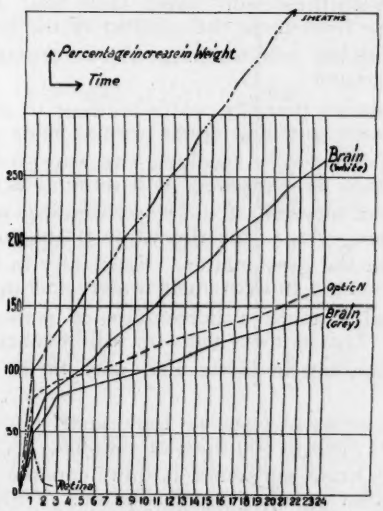


DIAGRAM II.

In every experiment we took two pieces of the tissue and kept them under the same conditions. The figures in the next schedule are averages, given in such a way that every figure, which was used for the calculation of an average was in itself already an average of two experiments. The numbers shown in this schedule, therefore, are the averages of at least sixteen experiments.

SCHEDULE I

	n	n/10	n/100	n/1000	H_2O	n	n/10	n/100	n/1000	H_2O
Hydrochlorid acid ...	+15	+38	+70	+ 90	—	+24	+45	+60	+99	—
Sulphuric acid ...	+18	+20	+29	+ 40	+264	± 0	+ 4	+17	+38	+135
Nitric acid	+27	+69	+82	+100	—	+12	+55	+49	+36	—
Acetic acid	+25	+18	+43	+128	—	+23	+24	+29	+74	—
Lactic acid	+94	+51	+90	+225	—	+76	+71	+89	+88	—
Citric acid	+92	+25	+40	+ 97	—	+56	+63	+84	+98	—

In the schedule the left series of figures shows the percentage of weight increase due to water-binding of the white brain matter, and the right series of figures shows the same of the grey matter. We were satisfied with the results given from four different concentrations, because from these the conduct of the brain in acid was quite clear. The last column shows the percentage of increase in weight in pure water.

Schedule I shows that the water-binding in acid is less than that in pure water, and that equal normal acids show a different water-binding. Generally speaking the water-binding is greater in inorganic acids as compared with mineral acids and furthermore, there is an increase of the water-binding when the concentration decreases. As a rule the white brain substance absorbs more water than the grey matter. Especially in lactic acid is the white matter inclined to take much water, and in a concentration of 0·001 normal it takes a percentage of water which exceeds 200 per cent. This is even more in still weaker concentrations. This specific conduct in lactic acid may be of some biological importance.

In a second series of tests we took small pieces of brain substance and kept them in solutions of alkalies. Schedule II shows, that in alkalies brain substance is quite capable of taking water. Furthermore, that in equinormal solutions of alkalies the water-binding is shown differently, and that the quantity of water-bound

increases with increasing concentrations. Here again the white brain matter shows a greater water-binding than the grey. In normal concentrations the water-binding was so great that it could be no longer accurately measured. This is expressed in the schedule by a line, showing that it was no longer possible to weigh the small pieces of brain substance without losing part of them.

SCHEDULE II

	n	n/10	n/100	n/1000	H_2O	n	n/10	n/100	n/1000	H_2O
NaOH ...	—	+270	+213	+107	—	—	+184	+ 94	+ 35	—
KOH ...	—	+264	+244	+186	+264	—	+165	+ 65	+ 62	+135
NH_3 ...	—	+287	+297	+280	—	—	+188	+129	+ 81	—

After this we made some tests in solutions of salt. In doing so we first took a series of cations and after that a series of anions. Only the latter are shown in the form of a schedule, as the former gave the same results. The results are shown in schedule III.

SCHEDULE III

	m	m'10	m/100	m/1000	H_2O	m	m/10	m/100	m/1000	H_2O
Na-cyanide ...	+20	+53	+114	+104	—	+ 3	+29	+68	+69	—
Na-rhodanide	+34	+38	+81	+113	—	+29	+45	+54	+79	—
Na-nitrate ...	+33	+44	+86	+231	—	+31	+37	+51	+97	—
Na-iodide ...	+39	+35	+80	+ 88	+264	+29	+30	+69	+80	+135
Na-bromide...	+30	+26	+51	+ 53	—	+22	+28	+62	+57	—
Na-tartrate ...	- 4	+29	+76	+ 85	—	- 8	+17	+51	+67	—
Na-phosphate	- 3	+12	+59	+120	—	± 0	+15	+55	+61	—
Na-sulphate	- 2	+33	+37	+ 70	—	- 3	+24	+56	+68	—
Na-citrate ...	- 7	+15	+72	+ 92	—	- 9	+25	+56	+78	—

From this it is evident, that in solutions of salt the water-binding from pure water is checked, and that proportionately to their concentrations. This checking influence of salt solutions is greater in the grey brain matter than in the white.

Equimolar salt solutions show a different influence. It is possible to arrange a series according to Hofmeister. This series shows different figures for white and grey brain matter respectively. Though the figures for white brain substance are not quite similar to those of the optic nerve they very much resemble these. Nearly the same results were obtained with anelectrolytes. Anelectrolytes also check the water-binding according to the concentration. Equimolar solutions show a different influence, and in these cases checking is greater for the grey brain matter than for the white.

From these series of experiments we may conclude, as could be expected, that the white and grey brain matters are capable of binding water, and that this water-binding is a real " Quellung." The capacity for water-binding is considerable; it is larger in the white brain matter than in the grey, and the capacity of the white brain matter is almost equal to that of the optic nerve. One might say that in these experiments the relation between the optic nerve and the white brain matter is once more shown.

For the purpose of the measuring the intensity of the water-binding we have made use of solutions of acetone and urea, as we did in our former paper. Schedules V and VI show that the water-binding out of acetone is small, even in a concentration as high as 15 per cent. after one hour. The same can be said of urea.

SCHEDULE IV

	m	m/10	m/100	m/1000	H_2O	m	m/10	m/100	m/1000	H_2O
Urea	+172	+125	+130	+123	+264	+103	+125	+113	+116	—
Glucose ...	+154	+147	+177	+215	—	+36	+ 71	+109	+124	+135 —
Saccharose ...	+ 23	+ 41	+ 67	+112	—	+ 3	+ 46	+ 53	—	—

SCHEDULE V

Concentration of Acetone

Time in minutes	10 °/o	20 °/o	30 °/o	40 °/o	50 °/o	10 °/o	20 °/o	30 °/o	40 °/o	50 °/o
1 minute	+ 6	+ 6	+ 6	+ 4	+ 3	+ 5	+ 6	+ 4	+ 6	+ 2
5 minutes	+ 6	+ 6	+ 4	+ 3	+ 4	+ 8	+ 5	+ 4	+ 4	+ 4
10 minutes	+ 9	+10	+ 7	+10	+ 5	+10	+10	+ 8	+ 7	+ 7
60 minutes	+29	+23	+16	+11	+ 8	+16	+20	+17	+14	+12

SCHEDULE VI

Concentration of Urea

Time in minutes	10 %	20 %	30 %	40 %	50 %	10 %	20 %	30 %	40 %	50 %
1 minute	+ 6	+ 3	+ 3	+ 6	+ 3	+ 2	+ 2	+ 2	+ 2	+ 4
5 minutes	+ 4	+ 6	+ 7	+ 6	+ 6	+ 6	+ 5	+ 4	+ 5	+ 5
10 minutes	+12	+11	+15	+11	− 9	+ 9	+ 7	+ 5	+ 7	± 0
60 minutes	+22	+22	+29	+14	− 12	+10	+13	+ 9	+ 4	− 2

SCHEDULE VII

Time in minutes	75 % Acetone		100 % Acetone		75 % Acetone		100 % Acetone	
	Loss of water	Percentage of water.	Loss of water	Percentage of water	Loss of water	Percentage of water	Loss of water	Percentage of water
1 minute	± 0	68	− 1	67	+ 2	75	− 9	76
5 minutes	± 0	67	− 5	69	+ 1	73	−16	71
10 minutes	− 2	68	− 7	70	+ 1	72	−42	74
60 minutes	− 3	70	−27	68.5	+ 1	74	−49	74

In the next series of experiments we put pieces of white and grey brain matter in concentrations of 75 per cent. and 100 per cent. acetone and measured their water-percentages for a time. These are shown in schedule VII. From this schedule it is quite clear that in the space of one hour pure acetone is capable of extracting from the white brain substance 27 per cent. of its water content. It must be borne in mind that normally the white brain substance has a water-percentage of about 68/69 per cent. In experimenting similarly with grey brain matter, 49 per cent. is lost, while the normal water-percentage is 74 per cent. The water-binding of the white brain substance appears to be almost similar to that of the optic nerve, both in manner of binding and in quantitative conditions.

Furthermore we have measured the change in the specific weights, as was done formerly with the other tissues of the eye. We found that in using white and grey brain matter, the specific weight increases gradually and slowly when the water-percentage is diminished. In our paper on the " Water-binding of the Optic Nerve and its Sheath " Diagram I shows the relations between retina and optic nerve in this respect. From this diagram it

appears clearly that the specific weight of the optic nerve is a little less than of the retina; but if the water-percentage of the optic nerve decreases the specific weight increases in a straight line. In the retina this increase is very rapid and great: this is shown in the curve. If we try to draw our results for white and grey brain matter in this diagram, we get two lines, which are almost exactly similar. The increase amounts from 1·05 to 1·1 when the water-percentage is decreased from 74 rasp. 68 to 0. From this it is quite clear that the water-binding of the optic nerve must be similar to that of the brain.

Finally we endeavoured to measure the rapidity of the loss of water on desiccation. Figure 2 shows the results. The ordinates show the percentage of loss of water, the abscissae the time in hours. Diagram II shows 5 curves representing the loss of water in relation to the time of desiccation at the temperature of 68·6° F. of the sheath of the optic nerve, white brain matter, grey brain and retina. This series shows directly an increase in rapidity of loss of water. Diagram II shows that the intensity of water-binding is smallest in the sheath of the optic nerve and brain matter, optic nerve and retina. This series shows directly an increase in rapidity of loss of water. Diagram II shows that the intensity of water-binding is smallest in the sheath of the optic nerve, largest in the retina and almost equal in the optic nerve and brain matter. The percentage of water shows a quite reverse figure. The water-percentage is largest in retina, less in white brain matter, optic nerve, grey brain matter and sheath of the optic nerve.

Summarising one may say that brains show the capacity of water-binding by "Quellung." The capacity, intensity and form of the water-binding are similar to those of the optic nerve.

The optic nerve is more closely connected with the white brain matter than with the grey. In regard to the pathogenesis of the papilloedema, our experiments show that one may expect that changes in the conditions which have some influence upon the brain matters may also show changes of the optic nerve. In other words, it can be expected that by increase of the power of water-binding a condition can be created which appears as a papilloedema, while at the same time the brain matters show a condition of swelling.

ATROPHIC RECESSION OF THE LAMINA CRIBROSA

BY

A. CHRISTIE REID

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MY apology for this paper is that several points in the pathogenesis of a condition that is not yet universally recognized or even acknowledged are still obscure.

The condition I have in view is one in which, without any demonstrable increase of tension and with the minimum of interference with the nerve elements passing through it the lamina is "receded," giving a hollowed-out appearance indistinguishable from glaucomatous cupping.

In the living subject this is accompanied by a wider exposure of the lamina of a measurable amount, as has been so well impressed on us by Mr. Ransom Pickard in his numerous papers.

That there may be such a condition apart from any increase of intra-ocular pressure should not be regarded as a physical impossibility. But on showing a case of this nature to a recent meeting I was faced with two curious objections (1) that the tissues of the optic nerve were absolutely incompressible, in the same way as any fluid is incompressible in a physical sense. This objection comes from a confusion of ideas. All tissues of the body are compressible because ultimately all are exposed to the compressible air that surrounds it. In other words we are not dealing with any tissue enclosed in an absolutely and permanently rigid envelope. All our so-called "rigid envelopes" can yield to pressure if time is given, because they are also living structures that can undergo changes in their structure and chemical composition. That this is true of the coats of the eyeball is proved by the quadrate eye of old untreated glaucoma.

(2) It was also objected that the pressures behind and in front of the lamina must be equal because we have there a state of equilibrium.

But the walls of a golf ball are in equilibrium with a centre pressure so much in excess that we know the risk of cutting into it. So with a tennis ball to a less degree. Equilibrium in these cases depends on three factors, the inside and outside pressures and the rigidity of the wall between them.

So with the eyeball; the rigidity of the wall is the factor that can maintain a position of equilibrium between the intra-ocular and the atmospheric or the tissue pressure, according to which is in contact with the part concerned.

In the case of the lamina it is the tissue pressure of the optic

nerve behind it, though this is of course in indirect communication through the body with the outer air.

My contention in showing the case—a lady, aged 70 years, tension decidedly subnormal, 26 McLean, with obvious and deep cupping and wide exposure of laminae, vision 6/6, fields to 3/1000 practically full, and no history of attacks suggesting raised tension—was to link up in theory views still somewhat at variance on the pathogenesis of a condition not in itself likely to benefit from measures directed against raised intra-ocular pressure. In fact I go so far as to say that many needless and useless operations are to-day being done on these cases from the mistaken idea that at some time or other they have had, (though this has never been demonstrated in a given case) or will certainly have, raised tension that will do damage.

I avoid the term cavernous atrophy as if it were an adequate explanation of what may concern the lamina mainly or only.

There may or may not be an accompanying cavernous atrophy but the essential feature is a yielding of the fibrous tissue of the lamina.

Now why should this yield? Let us think again of the tennis ball. If we thin down a disc on its surface sufficiently we get a small blister—as in a motor tyre. In exactly the same way the only factor required to produce a recession of the lamina, pressure before and behind remaining the same, is a thinning or weakening of its fibres.

The possibility and probability of this happening is at once evident when one realizes that the lamina has a blood supply of its own.

This has been demonstrated by Lagrange and Beauvieux in a paper at the meeting of the French Ophthalmological Society in 1924, which I had the pleasure of hearing. Referring to previous work they pointed out that it was impossible to inject the lamina post-mortem from the central artery of the retina. On so injecting, the parts of the nerve behind and in front of the lamina took the fluid. But on injecting the ophthalmic artery the lamina also is injected *via* the circle of Haller (or Zinn).

The sections show thus the absolute independence of the central artery and its non-participation in the irrigation of the lamina which depends, we repeat it, exclusively on the arterial circle of Haller.[1]

This circulation too seems to be terminal in character and no anastomosis with that of the central artery occurs.

The nutrition then of the fibrous lamina would be gravely compromised by any interference with its blood-supply coming from this circle; perhaps too that of the nerve elements traversing it, though, owing to the thinness of the lamina, it is conceivable that the supply from the central artery before and behind might be sufficient for these. I need not strain anatomical points however. The late Col. Elliot's paper should be consulted.[2]

Now one has only to suppose a localized narrowing of the circling vessels to give the exact condition required for a weakening of the lamina.

To demonstrate this may be impossible, but the subjects of this recession are usually past middle age and arterio-sclerosis can be demonstrated elsewhere.

The sclerosis may also involve the arterial supply to the nerve elements and so give a veritable optic atrophy. This may or may not be of cavernous type.

The essential thing to bear in mind is that such recession and even such atrophy may occur independent of any rise in intra-ocular pressure, and the additional factor of raised pressure is no added difficulty as Mr. Pickard supposes, when he says "It is difficult to see how it (cavernous atrophy) can arise in cases of increased tension." Lagrange's paper shows how it may and does.

We may then envisage three conditions that may combine or for a time at least remain separate and all three may occur independently of increased intra-ocular tension (*a*) primary atrophy of the nerve elements (*b*) cavernous atrophy involving both nerve and supporting tissue (*c*) atrophic recession of the lamina.

No conception of retraction is needed to explain (*c*) nor has retracting tissue ever been demonstrated in the nerve in these cases; on the contrary the changes have been of the nature of atrophy. In this event the picture is a combined one of (*b*) and (*c*), *i.e.*, with involvement of the nerve elements.

The term "atrophic recession" seems then to be adequate to express a condition occasionally met with, of cupping without involvement of the nerve elements and without begging an increase of pressure that cannot be demonstrated. Such a recession is in our clinical experience likely to be accompanied by an atrophy first of the glial elements in front of the lamina, so leading to a wider exposure. In Elliot's view this is an effect of pressure (*loc. cit.* p. 312) but in the following page he agrees with Fuchs that the problem "is not the simple one of the physical effects of pressure alone."

What then may be the other factor of importance? May it not be the blood supply of the glia (central artery) and of the lamina (circle of Haller)?

It is because sufficient emphasis has not been laid on this that I have ventured to correlate these few ideas.

REFERENCES

1. *Trans. of the French Soc.*, p. 465, 1924.
2. "On the yielding of the optic nerve head in glaucoma." *Brit. Jl. of Ophthal.*, p. 307, 1921.
3. *Brit. Jl. of Ophthal.*, p. 332, 1931.

FIBROCYSTIC DISEASE OF THE FRONTAL BONE (PAGET'S OSTEITIS)

BY

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THE case which is briefly recorded here is of interest both from a surgical and pathological point of view. A Hindu girl aged 20 years presented herself at hospital on March 11, 1935, with a diffuse protuberance of the left frontal region, a soft cystic tumour projecting from beneath the left supra-orbital rim, and proptosis of left eye. She said that the left side of the brow had become more prominent gradually during the last ten years. She maintained that the swelling of the eye had only become really marked within the week. The globe was pushed downwards. Its movements were not impaired except in an upward direction, and the vision was normal. The photograph gives a better idea of the condition than further description (Fig. 1). The fluctuating tumour was not pulsatile; mucocele was anticipated and it was explored. Serosanguineous fluid was obtained. An X-ray picture revealed a " nigger wool " skull. The reduced X-ray positives give an idea of the condition (Fig. 3). The proptosis required attention and a tarsorrhaphy was done, subsequent to the aspiration of 250 c.c. of sero-sanguineous fluid from the cyst. It filled again. It was decided to cut down on the cyst through the left brow, remove a portion of the frontal bone, drain the cyst from the temporal side and let the skin fall in. This was carried out. The cyst was lined for the most part with a smooth serous membrane. It roughly corresponded to what might have been an expanded frontal sinus, but presumably was not. The periosteum at the orbital rim was continuous with the membranous cyst wall or capsule which intervened between orbital contents and the cyst cavity. The roof of the cyst was somewhat granular to the examining finger and inclined to bleed. With fingers in the cystic cavity and the thumb on the frontal region one felt a thinned out edge at the orbital rim, but three-fourths of an inch back of this the bone was an inch thick. The periosteum was stripped from the orbital rim which was then cut back for about an inch. It cut readily with a scalpel and was of a gritty consistency like cuttlefish bone. The cavity was drained and the periosteal flap and skin allowed to fall in.

A photograph a year later (Fig. 2) shows that the large cavity had filled up and become lined throughout with skin leaving a

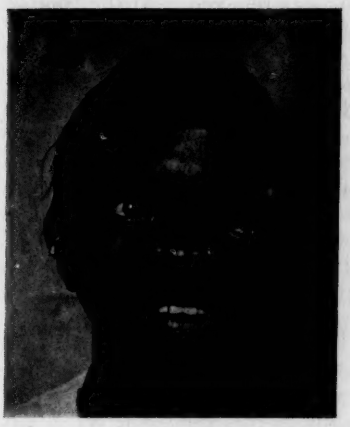

FIG. 1.

Showing the appearances on March 14, 1935.

FIG. 2.

Showing the appearances on April 18, 1936.

FIG. 3.

depression in the superior temporal quadrant of the orbit of relatively small size. The proptosis was relieved. At the time of operation suitable portions of the bony tissue were fixed for histopathological examination.

The X-ray appearances of the skull and the state of the bone at the time of operation suggested a diagnosis of Paget's osteitis with cyst formation, or some fibro-cystic condition of bone related to the osteitis fibrosa group. Further investigation favoured a Paget's osteitis although certain features are not in accord with what may be regarded as the accepted picture. Other points in the patient's record may be summarised briefly.

General.—The patient appeared to be in good health and was nursing a healthy baby a few months old. A year later they were both still in excellent condition.

X-ray examination.—With the exception of the changes in the skull the skeleton appeared normal in every respect. A complete examination was made on two occasions, March, 1935, and April, 1936. No obvious change in the skull at this time, nor yet in any other part of the skeleton.

Blood chemistry and urine.—Calcium 12.6 mgs. per 100 c.c. (on two occasions). Inorganic phosphate 3.45 mgs. per 100 c.c. serum. No Bence Jones protein or albumen in urine. The blood picture showed a definite lymphocytosis in March, 1935, but was within normal limits a year later.

The percentage of haemoglobin was 70 and 60 respectively on these occasions.

Histopathology.—Report by Dr. T. Bhaskara Menon, Pathologist, Stanley Medical School, Madras:—

"There is an invasion of the bone by a fibroblastic tissue, which shows here and there a few osteoblasts. There is absorption of the lamellae and rarefaction. The fibroblastic invasion is similar to that found in osteitis fibrosa cystica. The appearances vary somewhat in different portions of the tissue. In one preparation, the bony lamellae are separated by fibrous tissue showing well marked fibrocytes with collagen fibres. In places the fibrous tissue appears to be hyaline, the blood vessels few. Rarefaction is met with in some places. Another preparation shows rarefied bony lamellae in one area, with fibro-vascular tissue in the adjoining one. The fibro-vascular tissue shows areas of haemorrhage and perivascular infiltration with lymphoid and mononuclear cells. There are numerous fibroblasts besides well formed fibrous tissue. In a third the fibro-vascular inflammation of bone seems to be the predominant change. In addition there are extensive areas of haemorrhage."

The histopathology is consistent with the view that this is an instance of a localised fibrocystic disease of bone, falling within

the group in which there is no change in the calcium and phosphate metabolism, in contradistinction to the more generalised or diffuse fibrocystic disease with multiple bone cysts, hyperparathyroidism, altered calcium and phosphorous values in the blood stream, the former raised, the latter lowered. In view of modern work on the " osteitis fibrosa " group it might be better perhaps to avoid the names of Paget and von Recklinghausen until with improved classification of bone diseases, they can be attached to definite types without adding to the confusion which even now exists.

ACUTE DACRYOADENITIS DUE TO THE MORAX-AXENFELD DIPLOBACILLUS

BY

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IN July, 1936, Dr. N. Pillai drew our attention to a run of three cases of acute dacryoadenitis which he had seen in the out-patient's department of this hospital within a few weeks and asked our opinion as to their nature. In two of them a few small subconjunctival beads of pus showed over the region of the accessory gland; otherwise they all subsided without suppuration. The condition is ordinarily very uncommon, and as the aetiology was in doubt it was determined to make cultivations from the enlarged preauricular gland of the third case on a variety of media which usually favour the growth of the conjunctival flora. The media were those found most satisfactory by Dr. B. P. B. Naidu, of the Medical Research Department of the Government of India, when investigating the normal and pathogenic conjunctival flora in this hospital in 1935-36 while collaborating in trachoma research.

Short clinical note.—The patient, a Hindu boy, aged 19 years, first felt pain at the right inner canthus on July 26, 1936, five days before admission. Next morning his upper lid was red, swollen and tender. He had photophobia and lacrymation, tenderness in front of the ear and slight fever. On coming to hospital, there was chemosis of the bulbar conjunctiva, swelling in the superior temporal quadrant of the orbit with pain and tenderness. The swelling projected into the conjunctival fornix in the superior temporal region, giving the impression of an acute adenitis of the whole gland. The accessory gland was visibly inflamed. The cornea was clear. There was no discharge. It is regrettable that smears and cultures were not made from the

conjunctiva in any of the cases. The right preauricular gland was enlarged and tender. On admission, the temperature was normal, but rose to 100° the following day, then fell to normal and did not rise again. He was treated by hot fomentations. On the third day—seventh day of disease—the preauricular gland was punctured with the usual precautions to avoid organismal contaminations. A needle on a syringe containing about half a cubic centimetre of broth was introduced, and the point of the needle moved about in the gland, slight positive and negative pressure being alternately exerted. The gland pulp thus obtained in broth was planted on blood agar, serum agar and Soparkar's medium (for composition see *Indian Jl. of Med. Research,* Vol. VI, page 418), and yielded in all a pure culture of the diplobacillus of Morax and Axenfeld.

This does not necessarily indicate that the other two cases were due to the same organism, and the reader may well wonder why the other cases were not investigated in the same way, or, why the preauricular gland was punctured rather than the lacrymal gland. The reason is that we did not take the aetiological problem seriously till the third case was well advanced. It occurred to us then, that there was a possibility of an epidemic of acute dacryoadenitis and by this time the preauricular gland was easier to deal with. It may be objected that even in the one case investigated it is not proven that the organism isolated from the preauricular gland was necessarily the aetiological agent of the dacryoadenitis. This is of course true and theoretically one might obtain such a common inhabitant of the conjunctiva as the Morax-Axenfeld bacillus, from an apparently normal preauricular gland, were it practicable to obtain gland pulp. There is however a reasonable probability that the cause of the dacryoadenitis in one case of the three—and possibly in all three cases—was the bacillus of Morax-Axenfeld.

ANGIOGLIOSIS RETINAE WITH REPORT OF TWO CASES

BY

IDA CZUKRÁSZ, M.D.

(FROM THE UNIVERSITY EYE CLINIC, DEBRECEN, UNDER THE CHARGE OF PROFESSOR A. KREIKER)

v. HIPPEL first described angiogliosis retinae which is a part of the group-complex tumours of ectodermal origin found in the parenchymatous organs. In a fourth of the cases reported with cystic angiomas of the brain, spinal cord, tonsils, kidneys, pancreas,

ovaries, spleen, testicles, suprarenals and epidydimi glands, angiogliosis retinae was combined. Several cases have been reported establishing the recessive hereditary characteristic. Occasionally in members of the same family manifestations of this group-complex are evidenced by the presence of various parenchymatous tumours and angiogliosis retinae. Rochat's report suggests that this disease is transmitted through the female. It occurs most frequently in males.

The disease usually appears in childhood, but often adults present themselves to the ophthalmologist with loss of sight or pain.

Brand classifies four stages:—

(1) The alteration of the fundus vessels—they become tortuous and enlarged—at times combined with the appearance of solitary or multiple tumour-knots.

(2) The presence of haemorrhages and degenerative spots in the retina.

(3) Development of glaucoma, retinal detachment and other complications.

(4) The total destruction of the eye.

Radium treatment is advised by Sallmann, Moller and Ulrick when the diagnosis is made early. Good results have been published with this treatment.

In the later stages when glaucoma or detachment are present, enucleation is indicated to relieve pain or for cosmetic purposes.

Often the diagnosis is not made until after histological examination. The symptoms of glaucoma, the presence of retinal detachment, the tumour-shade from transillumination with the greenish-yellow fundus reflex suggest the condition of glioma retinae.

The following two cases seem to be sufficiently rare to be recorded and were found among 100,000 patients of the University Eye-Clinic in Debrecen during the past 15 years. They belong to Brand's third classification, but we diagnosed them as glioma retinae and removed the eyes.

Upon sectioning the eye-balls we expected to find a heavy glial tumour, but were surprised to see a thin vermiform growth beneath the retina. We thought that we had found an as yet undescribed parasitic disease in the eye-ball. Though the neoplasm was larger than most of the published ones, we could not explain the pathological changes. Only later when the diagnosis of v. Hippel disease was made, were the cases clear.

The bulbs were fixed in formalin, horizontally sectioned, and 140 paraffin serial sections examined.

Case I

A boy aged 8 years lost the sight of the right eye two months before entering the hospital. The patient had no complaints referable to any other disease. The family history was negative.

Clinical Notes.—There was no light perception in the right eye and the veins of the upper lid were tortuous. Between the superior and external recti muscles, 7 mm. from the limbus a dark-blue area 6 by 8 mm. was visible through the sclera. The corneal surface was hazy and stippled. Tension: + 3. The anterior chamber was very shallow. The pupil was of maximum width. A greenish-yellow fundus reflex was seen with focal light. There

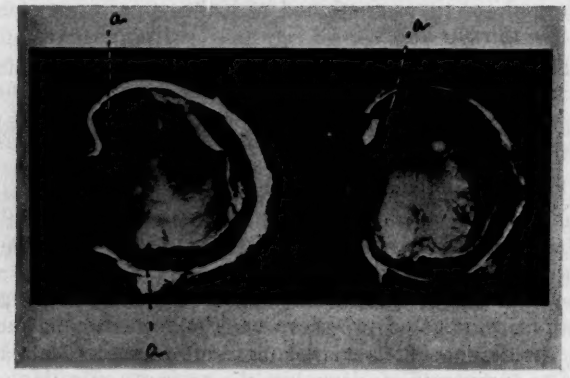

FIG. 1.

CASE I.—Photograph of the horizontally sectioned eye-ball. (*a*) Showing the neoplasm behind the ora serrata.

was a retinal detachment of the lower hemisphere bulging 3 to 4 D. into the vitreous. There were many retinal haemorrhages, some of them 1 to 2 disc diameters. Ophthalmoscopically no tumour was seen, but the Lange lamp test was positive.

Pathological findings.—Immediately posterior to the ora serrata behind the retina and adherent to it was a yellowish string of tumour tissue 2 mm. in diameter encircling the retina. There were numerous haemorrhages and yellow knots along the blood-vessels (Fig. 1).

We were astonished at the complete disorganisation of the retinal elements. The ganglion cells were destroyed. The nuclei of the inner nuclear layer and the cones and rods were partially necrotic. Glial tissue was abundant between the nerve and

FIG. 2.

Microphotograph of the tumour. (*a*) Tumour with spaces of different sizes. Some of them showing blood-vessels, some cholesterin crystals. (*b*) Disorganized retina, with cystic degeneration

FIG. 3.

(*a*) Place where the retina divided into two layers. (*b*) Large blood-vessels between the two layers.

Müller-fibres and among the remaining desolate neuroelements. The vessels were increased in number throughout the retina. They were enlarged and the perithelium thickened with hyaline degeneration. Amongst them glial-tissue, and pseudoxanthoma cells were present. In the latter we found fat, fuscin and cellular debris.

A tumour of 2 mm. in width was lying posterior to the ora serrata behind and connected with the retina. It was composed of hyaline degenerated tissue, cholesterin crystals and vessels. In the neoplasm were irregularly shaped spaces of various sizes, lined by endothelium and occasionally containing some blood. In some parts aneurysms were present (Fig. 2).

In some sections at the ora serrata the retina was indefinitely divided into two layers. We believed the former to be neuroepithelial structure and the latter to be of neuroelement. It was interesting to observe the tumour cells invading the two primary structures of the retina, and the choroid vessels. Some of the cells with well stained nuclei reminded us of embryonal neuroepithelium (Fig. 3).

Case II

Clinical Notes.—A boy of 11 years entered the hospital with pain and loss of vision in the left eye for six weeks. There was no light perception. The same marked glaucomatous signs were present as in Case 1. Retinal detachment was total. Several haemorrhages of different sizes and degenerative areas were present. Typical tumour findings were found with the diascleral light.

Pathological Findings.—A strange, reddish-grey, dychotomic, worm-shaped formation was seen in the upper part of the globe between the retina and the choroid (Fig. 4). This tumour was growing to the temporal edge of the optic disc and continued to the lower surface of the globe, and extended like a fan on to the adjoining retina. The structure of the retina was distorted here also. The nuclei of the first and third neurons were destroyed and only occasionally could we find a ganglion cell or cone and rod. Most pronounced was the disorganisation in the neuro-fibre layers as well as the layer of rods and cones. Vessels were increased in number and enlarged with a thick perithelium. The neural elements were replaced by neuroglial tissue and pseudoxanthoma cells. Mingled in the foamy-structure of these cells were considerable pigment granules (Fig. 5). Probably the conglomerations of fat in these cells created the ophthalmoscopic picture of the yellow degenerative spots. There were holes in the centre of these degenerations, representing the well-known cystic-degeneration of the retina.

FIG. 4.

CASE II.—Horizontally sectioned eye-ball. (*a*) Dychotomic vermiform growth. (*b*) Tumour masses partly along the vessels, partly underneath the retina. (*c*) Invasion of neoplasm to the optic nerve sheath.

FIG. 5.

Microphotograph of the destroyed retina. (*a*) Enlarged vessel containing blood. (*b*) Pseudoxanthoma cells.

FIG. 6.

Neoplasm invading the retina. (*a*) Glial tissue. (*b*) Enlarged retinal vessels. (*c*) Group of isolated vessels surrounded with pigment cells. (*d*) Capillaries and embryonal neuroepithelial cells in the vitreous.

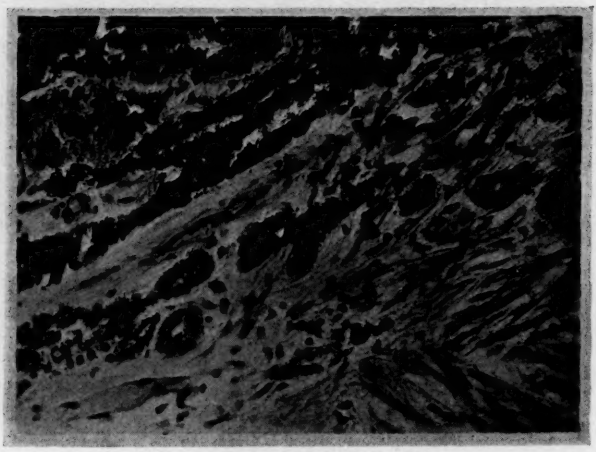

FIG. 7.

Pigment cells forming gland tubules.

The retina thickened gradually from behind forward and at the ora serrata the neoplasm invaded it (Fig. 6). Neuroglial tissue was also present in the tumour tissue. In the middle of the tumour mass was a large group of isolated vessels, consisting mostly of capillaries, surrounded with pigment cells. The structure of these pigment cells appeared similar to the structure of pigment epithelium. The structure of these cells could be ascertained only in those cells containing a slight amount of pigment. In some areas these cells appeared like gland tubules (Fig. 7). These

FIG. 8.

(*a*) Detached retina. (*b*) Tumour, directly connected with the pigment epithelium. (c) Pigment epithelium preforming neoplasm. (*d*) Phagocytes in the subretinal space.

formations and capillaries were present in the vitreous adjacent to the retina.

Cholesterin crystals and hyaline degeneration were seen in the growth that was lying free in the subretinal space.

The retina was detached up to the pars coeca plicata. The pigment epithelium layer was adherent to the choroid everywhere. Behind the pars plana coeca a branch of the tumour was directly connected with the pigment epithelium (Fig. 8). The cells of this area resembled the above described alveolar formations. A few millimetres behind this tumour was a tiny neoplasm of an entirely different structure. It consisted of 6 to 8 layers of honey-combed pigment cells.

The dychotomic growth at the disc invaded the lamina cribrosa and extended to the optic nerve sheath (Fig. 9). It was not an infiltrative growth and probably proceeded outwards for more favourable growing conditions. All of the above described elements we could find in this part of the tumour except pigment cells.

Phagocytes, wandering-cells and pigment cells were abundant in the subretinal space. We are of the opinion that the profound glaucoma in this case, which we cannot explain from the size

FIG. 9.

(*a*) Tumour between retina and choroid. (*b*) Neoplasm invasion of the optic nerve sheath.

of the tumour masses, was produced by a mechanical blockage of the angle of the anterior chamber by the wandering cells.

In these two cases we found a neoplasm, which combined with the glaucoma was responsible for the destruction of the retina. Though these tumours destroyed the function of the eyes and had expansive growth there was no evidence of infiltration nor metastases.

We feel that we can classify these tumours as hamartoma of the same type as the dysontogenetic tumours of the central nervous system.

Two opinions as to the origin of these tumours are found in the literature. Lindau, Möller, Rochat, Schuback, etc., claim that they have its origin primarily from blood-vessels (mesodermal). Meller and Marburg are of the opinion it is primarily a glial proliferation (ectodermal).

We are convinced that these tumours are ectodermal in origin, but we want to point out that we believe the initiating cause lies in the neuroepithelium. Probably it begins from embryonal rests, growing and breaking the junctions of the neuroepithelial cells. Upon such a basis we might explain the presence of glial tissue, epithelial cells of embryological character, and pigmentepithelial growth. In our minds, the presence of blood-vessels in the tumour is only of secondary appearance.

We feel that these two cases are convincing evidence that angiogliosis retinae is different from other angiomatous tumours of the parenchymatous organs, but it belongs, as Lindau says, to the systematic parenchymatous malformations.

LITERATURE

Karasek.—Angiomatosis Retinae. *Klin. Monatsbl. f. Augenheilk.*, Vol. XCV.

Junius.—Zur Frage der Angiomatosis Retinae. Ergebnisse und Folgerungen. *Klin. Monatsbl. f. Augenheilk.*, Vol. XCI.

Glinsberg, S., and Spiro, G.—Ueber Angiomatosis Retinae. *Arch. f. Ophthal.*, Vol. LXXXVIII.

Berblinger.—Zur Auffassung von der sogenannten v. Hippelschen Krankheit der Netzhaut. *Arch. f. Ophthal.*, Vol. CX.

Dtrói.—Weitere Angaben zur Entwickelung der Angiomatosis Retinae. *Klin. Monatsbl. f. Augenheilk.*, Vol. LXXI.

Rochat.—Familiäre Angiomatosis Retinae und Kleinhirnangiom. *Klin. Monatsbl. f. Augenheilk.*, Vol. LXXVIII.

Lindau.—Zur Frage der Angiomatosis Retinae und ihrer Hirnkomplikationen. *Zentralbl. f. Ophthal.*, Vol. XIX.

——— Studien ueber Kleinhirncysten. *Zentralbl. f. Ophthal.*, Vol. XVII.

v. Hippel, E.—Noch einmal zur Angiomatosis Retinae. *Arch. f. Ophthal.*, Vol. CXVIII.

Heine.—Ueber Angiogliosis Retinae und Hirntumor. *Zeitschr. f. Augenheilk.*, Vol. LI.

Marchesani.—Anatomie der Angiomatosis Retinae und Retinitis exudativa. *Arch. f. Ophthal.*, Vol. CIII.

Carr-Stallard.—A case of angiomatosis retinae. *Brit. Jl. of Ophthal.*, Vol. XVII.

Niccol-Moore.—A case of angiomatosis retinae. *Brit. Jl. of Ophthal.*, Vol XVIII.

v. Hippel, E.—Angiomatosis Retinae und Retinitis exudativa (Coats). *Arch. f. Ophthal.*, Vol. CXXVII.

OPHTHALMIC TREPHINE SCISSORS*

BY

J. EDWARD MARTIN

CLIFTON

I DESIGNED these scissors to facilitate the dissection of the flap before trephining, and I find them very comfortable in manipulation. The instrument has been made for me by Messrs. John Weiss and Son. The object of the design is to diminish flexion of the operator's wrist and thus avoid impairment of the finer movements of the hand.

* Reprinted from the *Brit. Med. Jl.*, Vol. I, p. 223, 1937.

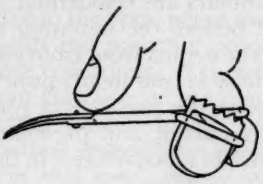

FIG. 1.—Cutting.

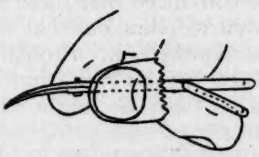

FIG. 2.—Blunt dissecting.

The details are—length : 3¼ inches; blades : curved on flat, with mean radius of curvature of 1 inch; points : fine, sharp, slightly ground off; joint : screw; bow for thumb : angled at 20 degrees from the straight, as shown in Fig. 1. A transverse section of the joint or shanks (when closed) would be quadrilateral, the flat sides assisting the grip in blunt dissection, as shown in Fig. 2.

ANNOTATION

National Ophthalmic Treatment Board, Annual Report, 1936

This report classifies the working of the Board under three general headings (1) The conduct of investigations into matters affecting sight. (2) The education of the public to a realisation of the importance of medical eye examination in all cases of suspected defect of sight. (3) The administration of the National Eye Service.

Under the first heading investigations have been conducted on the care of sight in industry. A simple form of questionnaire had been drawn up during the summer of 1935 and was sent to a number of firms employing labour. As a result more than 400 firms from all parts of the country replied. These replies were classified and then subjected to statistical analysis, and the results were published as a special report in July, 1936.

For three successive years analyses have been made of the findings of a series of examinations of the eyes of patients who had obtained treatment through the medium of the National Eye Service. The findings of the three years are shown in tabular form. " The agreement in the findings of these three analyses is unquestionable and noteworthy, the more noteworthy, since each year the number of ophthalmic medical practitioners sending in

returns has increased, so that each year's figures give what is virtually independent evidence."

" The broad fact elicited by these analyses of patients seen by ophthalmic medical practitioners is that out of 30,000 cases no fewer than 27·6 per cent. had ocular disturbances other than errors of refraction, and 7 per cent. had similar ocular disturbances without any errors of refraction; altogether 34·6 per cent., or more than one-third of the patients had defects or diseases apart from errors of focus."

As a contrast to these figures the Board has been able to obtain those of certain of the approved societies, which are " in effect the findings or diagnoses of the sight-testing opticians."

It appears that only 3 per cent. of those of their members who took ophthalmic benefit through sight-testing opticians were referred for medical examination.

The Board's Annual Report insists on the fact that this discrepancy between the percentages of 34·6 and 3 per cent. is much too wide to be bridged. Attempts have, of course, been made to whittle away their significance, and the report discusses at some length a recent paper by an apologist for the sight-testing opticians which deals with these analyses. It says that " the positiveness of the apologist destroys his case. Where the trained ophthalmic surgeon walks warily, this optician skips with alacrity." As an instance his statements on glaucoma are quoted. " Glaucoma, 0·62 per cent. Rarely would the presence of this disease be overlooked by the ophthalmic optician. . . . Although the disease is comparatively rare, the possibility of its presence and of its importance is so impressed upon the ophthalmic optician in his student days that he may be considered absolutely capable in detecting the condition and thus safe from the public point of view."

The second section of the report deals with education of the public, giving paragraphs referring to eyesight in industry, film propaganda, film projector service and Press articles. The last page of the report deals with the administration of the National Eye Service. This Service was established in 1929. The number of patients dealt with shows an increase of more than 32 per cent. as compared with the total number dealt with in 1935. The service now available in more than 400 places throughout the country has the assistance of nearly 900 specially appointed ophthalmic medical practitioners.

It is obvious that the Board is performing a public service to the community in more senses than one. We hope that the report will be studied by all concerned with the Public Health Acts. The attention of the Minister of Health should most certainly be drawn

to it; for it appears by a recent communication from the Board that he " proposes to submit to Parliament during the current session provisional regulations, entitled " Additional Benefits Amendment Regulations, 1937," which will give power to an " Approved Committee " composed of Approved Society representatives and representatives of sight-testing opticians to compile a list of sight-testing opticians without medical training or experience as " Recognised " as qualified to examine the sight of the insured population and to detect pathological conditions should they be present."

The Chairman of the Board points out that there is no occasion whatever for this Register of sight-testing opticians. Before these amending regulations are passed we think that the Minister should not only give his reasons for this change, so that the profession may know exactly what they are, but he should be asked how his Department can reconcile a difference between 3 and 34·6 per cent. The last question should provide an interesting problem for the statisticians of the Ministry.

If a body of unqualified persons be publicly recognised, by registration, as competent to test sight and to detect pathological lesions when present, it will be a bad thing both for the insured and the country. We do not question the ability of most of the sight-testing opticians in the purely optical side of the subject. It is on the pathological side that they are likely to fail; and if the idea that glaucoma is a comparatively rare disease is a fair sample of what they are taught, it makes us wonder how much value is to be placed on such teaching.

We call to mind a patient, a great sportsman, who came to us some years ago. He found that the glasses which he had procured from a branch of a well known firm of sight-testing opticians, made his eyes ache. The reason was obvious, the glasses were much too wide for his interpupillary distance. We asked him why he went to the shop in question. His reply was: " Oh, I saw ———, who used to play for Blackheath, going there one day and I thought what's good enough for him will do for me." Such a mentality is akin to that of Sydney Smith's hypothetical child who was told she might as well expect to please the Dean and Chapter of St. Paul's by stroking the dome; and reminds us of the elder Weller's sentiments about a good judge of cattle being a good judge of anything; an idea which, although Tony Weller didn't know it, is as old as the προβατογνώμων of Aeschylus.

It would seem that the public need protection from such ideas and their possible consequences, which may not always be forthcoming if these amending regulations are passed.

ABSTRACTS

I.—COLOUR VISION

Wright, W. D.—Recent work on colour vision—a review of papers published in 1935.*

The problems of colour-vision are being attacked by both physicists and physiologists along four broad fronts: (*a*) by the measurement of the colour vision characteristics of different types of observer; (*b*) by various psycho-physical experiments; (*c*) by examination of the structure and the chemistry of the retina; (*d*) by the measurement of retinal action potentials and the recording of nerve-impulses.

Heading (*a*) concerns the measurement of those characteristics of an observer's eye which determine the properties of his colour perception mechanism. These characteristics include the colour mixture functions and the hue and saturation discrimination data. The information regarding normal trichromats is now fairly complete, but there have been some wide gaps in our knowledge of the so-called " colour-blind " observers. Two papers published in 1935, one by Pitt[1] and the other by Trendelenberg and Schmidt[2], have helped considerably to fill in the gaps. Pitt's work was largely confined to dichromats, and his report provides very complete information for a relatively small number of observers. The luminosity curves, dichromatic coefficient curves, and hue discrimination curves are given for six protanopes and six deuteranopes. The two groups appear to be well defined and comparatively easy to distinguish from the trichromat by their reduced hue discrimination in the green-red part of the spectrum, and from one another by having very different luminosity curves. The data obtained have been used to subdivide the trichromatic colour triangle into a series of isochromatic zones, where any zone represents the locus of colours that, although seen by the trichromat as quite distinct, are indistinguishable to the dichromat when adjusted to the same intensity level. Introductory data on 3 anomalous trichromats are also given. The paper by Trendelenberg and Schmidt is in a sense complementary to that of Pitt, in that they made a simple test with Nagel's anomaloscope on a large number of observers (129 colour defective persons) and were able to subdivide them into the four broad groups of protanopes, deuteranopes, protanomalies and deuteranomalies. In addition to the relative distribution of the

* This article is reprinted by the kind permission of the Council of the Physical Society, from Reports on Progress in Physics, Vol. III, 1936, published by the Physical Society.

four groups, the important fact emerges that the two groups of anomalous trichromats are well-defined among themselves and do not grade into either the normal trichromat on the one side nor into the dichromat on the other. This is of very great theoretical interest as it appears to cut across the idea of any gradual evolution of colour vision, for on this basis a continuous distribution of observers from the monochromat through the dichromat to the trichromat, would be expected. The correct explanation must for the present remain a matter of speculation.

Section (*b*) includes all variety of experiments on flicker, adaptation, contrast, interaction between retinal areas, and so on. In a paper by Graham and Marajaria[3], curves are given showing the relation between the intensity of a patch of light and the time of exposure for the patch to be just visible, when viewed in the peripheral retina. As the area of the field is reduced, the curves tend to approximate to similar data obtained by measuring the electrical response from a single cell in the eye of *limulus*. It is concluded that the statistical distribution of the characteristics of a number of cells is responsible for the experimental curves and for their change with area. This conclusion is interesting, among other reasons, as an illustration of the part that statistical variations of sensitivity and response must play in visual perception, where large numbers of nerve endings are acting simultaneously.

Hecht and his collaborators have obtained results on the critical frequency of flicker and its variation with field size[4], and the results provide still further evidence of the separate activities of rods and cones. This group of workers have also been responsible for experimental and theoretical work on adaptation and intensity discrimination[5, 6], but experiments described by the present writer[7], do not support Hecht's photo-chemical theory of intensity discrimination. These experiments demonstrate that there is no direct connection between the concentration of the photo-chemical substance in the retina and the increase of a stimulus necessary to produce a just noticeable intensity difference.

Among other contributions that should be included under heading (*b*), some very suggestive work by Bouman[8] may be mentioned. Results on the electrical excitability of the eye have led to the conclusion that the origin of the excitation is not in the rods or cones, but in the nerve fibres either in the retina or in the optic nerve. He has found no evidence for two thresholds, and the level of excitability is determined by the state of adaptation. Flicker phenomena were observed and gave a fusion rate of 120 per sec.

Although little has been published during 1935 that might be included under heading (*c*), Tansley[9] has reported differences in the chemical constitution of the light and dark-adapted eye of both the frog and the rat, while Hosoya and Saito[10] have confirmed

the production of visual yellow from visual purple, and claim to have produced a visual white which they regard as a very pure visual yellow. It is obvious that all information about the constitution of the light-sensitive substances in the retina must have a very direct bearing on our ideas of the visual processes, but at the same time the difficulties of isolating sufficiently pure material in any but very small quantities, make this method of approach to the problem arduous, though attractive.

Some exhaustive work by Österberg[11] on the topography of the human retina, has added considerably to our knowledge of the relative distribution of the rods and cones. He found, for example, that the number of rods increases to a maximum at a distance of about 5-6 mm. from the fovea, and that the upper and nasal parts of the retina are better supplied with rods than the lower and temporal parts. The total number of receptors in the human retina was found to be between 110,000,000 and 125,000,000 rods and between 6,300,000 and 6,800,000 cones.

The work in heading (*d*) on retinal action potentials has been, perhaps, the most fruitful of any. The researches of Granit and his colleagues[12, 13], have resulted in a further analysis of the different components of the potential. The relation between the components for different conditions of adaptation has been examined and the increase of inhibition with light adaptation established. Two types of retinae have been found and named *E*-retinae and *I*-retinae, which are characterised by several distinctive features, of which the chief is the direction of the response when subject to intermittent stimuli. There is the possibility that *E*-retina, of which the cat's is an example, may correspond to rod-retinae, while *I*-retinae, as exemplified by the pigeon, may correspond to cone-retinae.

Graham and Hartline[14] have recorded the responses of single cells of *limulus*, but have found no variation in the type of response as the wave-length of the light stimulus was varied. Small variations in the luminosity curves of different nerve endings were, however, detected.

Graham, Kemp and Riggs[15] have experimented with a pigeon's eye (a colour-discriminating eye) and have recorded the responses, using both white and coloured stimuli. They found that the type of response for one wave-length could always be matched by the response for any other wave-length, when a suitable intensity was used. This result is in disagreement with similar work by Kohlrausch and Brossa[16], and no final conclusions can be arrived at yet, especially as the magnitude of the responses seems to have been rather small. The question is, however, of vital importance since, if any specific effect were found, it would be the first direct clue to the individual character of the different colour responses.

It is to be hoped that further work on these lines will be carried out.

In this review reference has been made to papers that seemed to the writer to be typical of the work in progress and it is in no sense an exhaustive survey. The bibliography includes only those papers directly referred to, and as these have been selected from a list of over 80 papers published in 1935, it will be appreciated that much work of value has had to pass unnoticed.

One point especially should emerge from this review, namely, the necessity for the close co-operation of physicists and physiologists, and the importance of each group of workers keeping in touch with the progress made in other fields.

REFERENCES

1. Pitt, F. H. G.—*Spe. Rep. Ser. Med. Res. Coun.*, London, 1935.
2. Trendelenberg, W., and Schmidt, J.—*S.B.* Preuss. Akad. Wiss, Vol. II, 1935.
3. Graham, C. H., and Marajaria, R.—*Amer. Jl. Physiol.*, Vol. CXIII, p. 299, 1935.
4. Hecht, S., Schlaer, S., and Smith, E. L.—*Quantitative Biology*, Vol III, p. 237, 1935.
5. Hecht, S., Haig, C., and Wald, G.—*Jl. gen. Physiol.*, Vol. XIX, p 321, 1935.
6. Hecht, S.—*Jl. gen. Physiol.*, Vol. XVIII, p. 767, 1935.
7. Wright, W. D.—*Jl. Physiol.*, Vol. LXXXIII, p. 466, 1935.
8. Bouman, H. D.—*Arch. néerl. Physiol.*, Vol. XX, p. 430, 1935.
9. Tansley, K.—*Jl. Physiol.*, Vol. LXXXIV, p. 34, 1935.
10. Hosoya, Y., and Saito, Z.—*Tohóku. Jl. exp. Med.*, Vol. XXVII, p. 172, 1935.
11. Österberg, G.—Thesis Copenhagen Univ., Nyt Nordisk Forlag, Copenhagen, 1935.
12. Granit, R., and Therman, P. O.—*Jl. Physiol.*, Vol. LXXXIII. p. 359, 1935.
13. Granit, R.—*Jl. Physiol.*, Vol. LXXXV, p. 421, 1935.
14. Graham, C. H., and Hartline, H. K.—*Jl. gen. Physiol.*, Vol. XVIII, p. 917, 1935.
15. Graham, C. H., Kemp, E. H., and Riggs, L. A.—*Jl. gen. Psychol.*, Vol. XIII, p. 275, 1935.
16. Kohlrausch, A., and Brossa, A.—*Arch. Physiol.*, Vol. CDXXI, 1914.

II.—MISCELLANEOUS

(1) **Manolesco, Lazaresco and Schmitzer (Bucharest).—Tumours about the sella turcica. (Contribution a l'étude des tumeurs de la region sellare).** *Rev. d'Oto.-Neuro.-Ophtal.*, September-October, 1936.

(1) **Manolesco, Lazaresco and Schmitzer** refer to reports which they have published in earlier numbers of this review; they have come to some conclusions in the matter of practice which they summarise as follows; in the case of a suspected tumour of the sellar region, it is absolutely necessary to examine the colour fields; and to examine the variations of light perception over the whole field. In all cases of oculomotor

paralysis the visual fields should be examined; where the diagnosis of a tumour in this region has been made, radiotherapy must be used; they have noted after radiotherapy a recovery of the visual fields by separate islands. The two cases which they record now, confirm these points.

HAROLD GRIMSDALE.

(2) **Pergola (Sassari).—Ocular manifestations in xeroderma pigmentosum. (Alterazioni oculari nello "xeroderma pigmentosum").** *Rass. Ital. d'Ottal.*, May-June, 1936.

(2) Xeroderma pigmentosum is a disease affecting primarily those parts of the body which are generally exposed; pigmented patches appear on the face, neck and hands; these patches often become the seat of malignant growth. **Pergola** has observed three cases, two brothers and a cousin, in whom the patches were noted within a few days of birth; it is often discovered after some acute exanthem; here there could be no question of its being due to such cause; it was noted that in these children the patches seemed to lessen in the summer and specially after sea bathing, and to reappear in the winter. The situation of the lesions would have suggested that the direct action of the sun and the ultra-violet rays were an important factor. The author thinks the disturbance to be due to some fault in the endocrine glands and especially in the thyroid.

HAROLD GRIMSDALE.

(3) **Thomson, Sydney (London). — Poikiloderma congenitale.** *Brit. Jl. of Dermat. and Syph.*, Vol. XLVIII, pp. 221-234, 1936.

(3) In this paper **Thomson** reports cases, and reviews the literature of a rare dermatological condition appearing in female infants as a pink swelling of buttocks, cheeks, later lateral and posterior aspects of the limbs, eyebrow areas, and external folds of the ears. Through the pink swellings gradually appears a telangiectatic network, with a few larger and whiter patches interspersed. Later, pigmentation replaces the vascular network, and a fine atrophy of the skin occurs in affected regions.

The cephalic contour is peculiar, the face having a pointed chin, so that the outline is an inverted triangle with apex below. In one case this disproportion simulated hydrocephaly, and another case, in fact microcephalic though with normal mentality, had the same triangular facial contour. One case had absent metacarpals and phalanges of the thumbs, while ulnae and radii were rudimentary.

There seems a tendency to associated ocular abnormalities. One of the author's cases has microcornea, high myopia, and partial optic atrophy. Lutz has described a case with definite thyroid

hypoplasia and double cataract. Bloch has described two somewhat typical cases with " juvenile cataract."

Six definite cases have been described in the literature, while three other cases are more doubtful. The first two cases were reported by the author in 1923. The syndrome is named by him "poikiloderma congenitale" from certain resemblances to Jacobi's disease, the poikiloderma atrophicans vasculare of adult life.

L. H. SAVIN.

(4) **Carroll, Frank D.—"Alcohol" amblyopia, pellagra polyneuritis, report of ten cases.** *Arch. of Ophthal.*, Vol. XVI, No. 6, December, 1936.

(4) Carroll's paper opens with a series of quotations from the literature which supports the view that so-called alcoholic polyneuritis and the alcoholic type of pellagra are in reality deficiency diseases and can be cured by a proper diet even if the alcohol is continued. He then reports 10 cases of " the clinical syndrome variously called tobacco-alcohol, tobacco or alcohol ambyopia which occurred in patients with the alcoholic type of pellagra or polyneuritis "; all smoked in moderation and all but one used cigarettes, but they were all heavy drinkers, consuming between 1 and 3 quarts of alcoholic liquor daily. In every case the diet had been inadequate and when this was rectified by a diet high in vitamin B and B^2 supplemented by liver extract injected intramuscularly improvement in vision occurred. A typical case was a 32 year old negro whose vision, failing for six months, had come down to 18/200 in the right eye and 10/200 in the left with a large caeco-central scotoma. He habitually drank 1 pint of whisky and smoked 4-5 cigars daily. His skin showed the linous characteristics of pellagra. After 35 days of dietetic treatment, vision in each eye was 20/40 and a year later was 20/20.

F. A. W-N.

(5) **Jeans, Blanchard and Zentmire (U.S.A.).—Dark adaptation and vitamin A.** *Jl. Amer. Med. Assoc.*, February 6, 1937.

(5) A new photometer and a technique for its use in the determination of ability to adapt to darkness has been developed. The intensity of the light with which the photometric readings are made is controlled by a rheostat, which is calibrated in terms of millifoot candles; this permits the results of one investigator to be compared with those of another, a procedure generally difficult for dark adaptation tests.

Evidence has been obtained for considering the test a useful means for detecting vitamin A deficiency.

A. F. MACCALLAN.

(6) **O'Day, Kevin.—A preliminary note on the presence of double cones and oil droplets in the retina of marsupials.** *Jl. of Anat.*, Vol. LXX, July, 1936.

(6) **Kevin O'Day's** work was carried out on the eyes of Dasyurus viverrinus whose habits are mainly nocturnal. Ophthalmoscopically, there was a well marked tapetum in the upper half of the fundus, but no direct evidence of a fovea, although the arrangement of the vessels suggested the demarcation of a central strip of retina. Histologically, the cones were found to be double and in their ellipsoid positions, to contain a globular mass which did not take Mallory's stain. An eye from another animal was fixed in osmic acid and sectioned in paraffin, when it was found that the globular mass was stained a dense black and was therefore presumably an oil droplet.

Examination of the retina of the ring-tailed opossum revealed the same conditions—*i.e.*, double cones and oil droplets.

F. A. W-N.

(7) **Denti (Milan).—Fundus lesions following severe injury to the thorax. (Lesioni del fondo oculare per gravi traumi toracici).** *Boll. d'Ocul.*, November, 1936.

(7) Fundus lesions following indirect violence are not common. **Denti** records two cases in which serious injury to the thorax by crushing, involving fracture of several ribs, was complicated by gross fundus changes. In both, the patient on recovering consciousness noticed defect in vision.

Both had much subconjunctival and subcutaneous haemorrhage of the face and neck. In both the margins of the optic discs were slightly indistinct; the vessels all engorged and tortuous, and the retinae slightly oedematous. In one eye the disc was pale from the first and vision was lost, in this eye, there were also white patches between the disc and the macula. In the second case vision at first became normal but soon failed to 6/18; at this level it remained.

The author thinks that the lesions are due to the disturbance of the circulation in the cranium following the sudden increase of pressure in the thoracic vessels from the compression.

HAROLD GRIMSDALE.

(8) **Nastri (Naples).—The general condition of the patient after ophthalmic operation. (Ricerche intorno alla fisiopatologia dell'operato nel campo oftalmologico).** *Boll. d'Ocul.*, October, 1936.

(8) It has been shown that after any large surgical interference the metabolism of the patient is altered; the signs have

been called the post-operative toxic syndrome; sometimes these are slight only, sometimes very evident. Roughly the intensity corresponds to the size of the operation. It was not to be expected that there would be much evidence of this state after operations on the eye, since these are for the most part small and short in execution.

Nastri has investigated the condition of the blood of patients after ophthalmic operations and finds that in most cases there is a slight reduction of chlorides and of calcium; these are changes similar to those which are found after the larger operations of general surgery, but are much smaller in extent corresponding to the extent of the operation.

HAROLD GRIMSDALE.

(9) **Rindello (Naples).—Experiments with lysozyme. (Ricerche sul Lysozym in rapporto ad alcune questioni interessanti l'oftalmologia).** *Boll. d'Ocul.*, November, 1936.

(9) Since Fleming in 1922 published his account of this bacterolytic body, which is found in many tissues, much has been written about it. **Rindello** has paid special attention to the behaviour of the xerosis bacillus in the presence of lysozyme. The bacillus is an almost constant habitant of the conjunctiva; he has found that tears, by their content of lysozyme, are able to dissolve the bacilli even in dilution. The bacilli in cases of xerosis seem even more vulnerable than those of the normal conjunctiva and the question is why they flourish in the presence of the tears. He finds that in these subjects the amount of ferment in the tears is smaller than normal but thinks that other factors enter also; he suggests diminution of the quantity of tears and of the contact of the tears with the conjunctiva as probable causes.

The author has tried to estimate the bacteriolytic power against the bacteria commonly found in the conjunctival sac, he has found it active against staphylococci and streptococci but inactive against diphtheria, pneumonia and anthrax bacilli, he has not found any advantage from the instillation of lysozyme, into the conjunctival sacs of rats suffering from xerosis from lack of vitamin A. The disease has run its usual course in spite of the instillations.

Nor has it had any success in cases of human trachoma in which xerosis was beginning.

HAROLD GRIMSDALE.

(10) **Magitot, A. (Paris).—Pathological Hypotony.) (Hypotensions oculaires pathologiques).** *Ann. d'Ocul.*, Vol. CLXXVIII, p. 785, 1936.

(10) **Magitot** gives a survey of hypotony occurring in malformations including myopia, after injuries, in retinal detachment, in local infections and toxaemias, in alterations of the composition

of the blood, in endocrine disturbances and in affections of the ocular sensory and vasomotor innervation.

HUMPHREY NEAME.

(11) **Fontana (Modena).—Weltman's reaction in ocular disease. (La siero coagulofiocculazione di Weltman in alcune affezioni oculari).** *Rass. Ital. d'Ottal.*, May-June, 1936.

Rubino (Padua).—Weltman's reaction in ocular disease. (La soglia elettrolitica in alcune affezioni oculari). *Rass. Ital. d'Ottal.*, July-August, 1936.

(11) **Fontana,** in his researches on the electrolytic threshold in various ocular diseases has found constantly a displacement of the KB to the left, a condition which Weltman takes to indicate inflammation. The author concludes that the alteration of the reaction may show a special change in the colloidal condition of the serum but cannot give any help in the diagnosis of ocular disease.

Rubino, on the other hand found the reaction in cases of cataract usually shifted to the right. He thinks that the shift to the left which is almost constant in glaucoma is due to the special state of the tissues and humours in that disease.

HAROLD GRIMSDALE.

(12) **Prister (Trieste).—A trap for malingerers. (Una trappola per simulatori).** *Boll. d'Ocul.*, July, 1936.

(12) In this test, coloured letters on a black ground are illuminated momentarily and shown to the examinee who is wearing red and green glasses. Since the image is illuminated only momentarily but persists for half a second, the man cannot tell by winking which eye receives the image. In a number of trials he is certain to make mistakes and to give himself away. The letters and their colour can be changed at will by the examiner.

HAROLD GRIMSDALE.

(13) **Jameson, P. Chalmers (Brooklyn).—Subconjunctival section of the ductules of the lacrimal gland as a cure for epiphora.** *Arch. of Ophthal.*, February, 1937.

(13) **Jameson's** operation has been devised as an alternative to extirpation of the lacrymal gland in cases of intractable epiphora. The latter, although a simple operation may be followed by thrombosis, pressure on the optic nerve, orbital haemorrhage and ptosis. Up to date, the author has performed his operation 6 times with complete success. The technique is as follows. Under local deep anaesthesia, an incision is made in the

conjunctival surface of the lid, adjacent to and slightly below the outer canthus. This must be large enough to admit Steven's scissors and allow free manipulations. The upper lid is everted, so that the fornix is put on the stretch, the scissors are then introduced into the incision and made to separate the conjunctiva from all the basic tissues of the upper fornix in its outer two-thirds, from the margin of the tarsal cartilage to the beginning of the bulbar conjunctiva. The conjunctiva being translucent, the points of the scissors should be visible throughout the operation, if they disappear from view it means that they are being passed too deeply, and the levator muscle may be injured. The after-treatment consists in the use of a compression bandage for a day or two and daily irrigation of the conjunctival sac. The primary opening in the conjunctiva remains patulous for 10-14 days and is useful for drainage. Cure is complete when this wound has closed.

F. A. W-N.

(14) **Reiger, H. (Vienna). — On the importance of choroidal changes for the aetiology of vitreous detachment. (Ueber die Bedeutung der Aderhautveränderungen für die Entstehung der Glaskörperabhebung.** *Arch. f. Ophthal.*, Vol. CXXXVI, p. 119.

(14) In his research on the aetiological importance of changes in the choroid, degenerative as well as inflammatory, in vitreous detachment **Reiger** examined 182 persons of all ages with simple myopia of all grades showing fundus changes.

He found that the incidence of vitreous detachment increases with the degree of the myopia and the age of the patients. This may be explained by the increase of fundus changes in these cases, and tables are given to show that there is a very close connection between these changes, essentially choroidal, and the age and myopia, and between the changes in the choroid (more so when at the posterior pole than at the periphery) and the vitreous detachment itself.

A study of the influence of the degree of the myopia alone, apart from the age, in cases of high anisometropia (over 4D. difference) gave no conclusive results, but where the vitreous detachment was unilateral it usually occurred in the eye with the higher myopia.

The evidence thus obtained affords no absolute proof of the importance of choroidal changes in the causation of vitreous detachment, but it receives support from a study of other diseases in which these conditions are associated, *viz.*, senile fundus changes, retinitis pigmentosa and inflammatory affections of the uvea.

In senile vitreous detachment (68 cases) a relatively large number of the patients showed only slight affection of the choroid; an analysis of the cases seemed to indicate that advanced disease of the vascular system must be present if vitreous detachment is to occur when the fundus changes are slight, but is not a necessary condition when the latter are more marked.

As regards retinitis pigmentosa the cases recorded, though few (14), suggest an aetiological connection between choroidal changes, especially at the posterior pole, and vitreous detachment.

This connection is more definite in inflammatory affections of the uvea, the risk of vitreous detachment increasing according to the age of the patient, the presence of myopia, and the duration and extent of the disease.

The results of examination over such a wide range of cases, it is claimed, demonstrate the great importance of choroidal changes in the causation of vitreous detachment.

As to the actual rôle which they play it is hinted that through them colloid-chemical changes in the fibrillar framework of the vitreous ensue; in this altered condition of the vitreous mechanical concussions, or vibrations, either through trauma or even rapid movements of the eyes, may act as the exciting cause.

THOS. SNOWBALL.

BOOK NOTICES

La Prophylaxie de la cécité en Roumanie. (The Prevention of Blindness in Roumania). By NICHOLAS BLATT. Pp. 53. Bucharest: Published by the International Association for the Prevention of Blindness. 1937.

This report embodies the reply prepared for Roumania by Dr. Blatt in response to the questionnaire sent out by the headquarters of the International Association in Paris, asking for information on the subject from the various countries included in the organisation. The report is arranged under the headings:—Statistics (number and causation of cases), legislation, financial resources, administration and propaganda.

The population of Roumania is approximately 19 millions and it is estimated that 0·72 per 1,000 are blind. An analysis of causes of blindness shows a high proportion of ophthalmia neonatorum, while syphilitic optic atrophy is also common. Interesting differences from conditions in this country are brought out by the inclusion of typhus, leprosy and pellagra in the list. Cataracts other than senile also show a high percentage and it is suggested

that this might be associated with endemic goitre and hypothyroidism in certain districts. Trachoma only accounts for a relatively small number of cases and the figures show that it is steadily diminishing. There are, however, a very large number of cases in which the cause cannot be ascertained, so that the figures given can be taken as a sample only.

The legal section is concerned with the vaccination laws governing venereal and other infections and midwives' acts concerning the obligatory use of Crédé's method. The report shows that the authorities in Roumania are fully alive to the importance of the work and are taking an active part.

I. M.

Diseases of the Eye. By EUGENE WOLFF. Pp. 225, Illustrations, 120. Cassell & Co. 1937. Price, 15s.

This textbook is written for students and practitioners. It is admirably illustrated with clear diagrams, figures, photographs of anatomical dissections and microphotographs of normal and pathological tissues. The author has attempted to compress into a relatively small space the essential and practical aspects of ophthalmology.

There are several parts which might have been dealt with a little more fully. For the general practitioner a more detailed description of ophthalmia neonatorum, its prevention and treatment could have been given with advantage. Under Parinaud's conjunctivitis no mention is made of the lesion produced by B.tularense and in describing tuberculous conjunctivitis the common cockscomb variety is omitted from the clinical account of the disease. In figure 4 cysts at the ora serrata are incorrectly labelled crypts and in figure 45, a visual field chart, the annular scotoma in retinitis pigmentosa is not a typical one as regards its situation.

In the treatment of entropion the author places injections of 80 per cent. alcohol before a skin-muscle operation after other therapeutic measures have failed. A number of ophthalmic surgeons would consider the skin muscle operation the safer and more reliable procedure of the two.

In the treatment of rodent ulcer radium has priority, but there is no mention of the contraindications to this treatment if there is bone involved in the growth.

There is much to be learnt from the illustrations and the book is admirably produced.

Medical Treatment of Cataract. By A. EDWARD DAVIS. Pp. 155. Philadelphia: F. A. Davis Company. 1937. Price, 3.00 dollars.

In the foreword of this book the author states: " Public opinion will finally compel every progressive ophthalmologist to employ

all preventive medical measures in such cases before resorting to operation. If such knowledge were carried to the general practitioner, and if early active treatment were established by the specialist, at least one-half (perhaps three-fourths) of all operations for senile cataract as now performed would be rendered unnecessary.

Chapters I to VI are expended on an historical survey of ophthalmology with particular regard to cataract; its aetiology and its associations with toxaemia from faulty metabolism, focal sepsis, eyestrain and endocrine disturbances; its diagnosis; symptoms; pathogenesis and spectroscopy.

In the chapters dealing with therapy the author describes at length his treatment by subcutaneous injections on alternate days of lens antigen, beginning with 0·5 c.c. in women and working up by 0·5 c.c. increases to 8 c.c. in women and 10 c.c. in men. During the injections potassium iodide is administered internally. This combination of therapeutic agents renders accurate assessment of the value of either one or the other an impossible task. The constituents of diet in relation to the treatment of cataract are also described at some length particularly in regard to vitamins, calcium, potassium, phosphorus, proteins and carbohydrates.

Dr. Burdon-Cooper has contributed the chapter entitled "Errors in refraction, and their influence on the production of incipient opacities in the lens and their therapeutic value in the treatment of cataract." To the reviewer this chapter was quite unconvincing.

In the descripton of other methods of non-operative treatment of cataract certain chemicals which are used as drops, and subconjunctival injections, medicines and subcutaneous injections, dionine, mercuric cyanide, potassium and sodium iodide, glycerine, boric acid and hormones such as thyroid extract, thyroxin, and gonadal extracts, radium, phakolysin and euphakin are mentioned.

Chapter XI is devoted to case reports and chapter XII to the experiences of other observers with favourable and unfavourable reports.

The subject is a difficult one and with a disease which in a number of cases may become arrested spontaneously or progress very slowly over a number of years without or in spite of treatment it is almost impossible to assess the true value of any particular therapeutic agent and even more so when several are employed simultaneously.

After reading this book one is left unconvinced. At the same time one realises that the author's advice with regard to the elimination of focal sepsis, the treatment of any co-incident general

medical disorder, dietetic adjustments and the correction of refractive errors is justifiable. It is probable that these counsels are practised by the majority of ophthalmologists.

The Medical Annual. Edited by H. LETHEBY TIDY and A. RENDLE SHORT. Pp. 704, Plates, LXVIII, Figures, 173. Bristol: Wright. London: Simpkin Marshall. 1937. Price, 20s.

This number of the Annual is characteristic of the high standard maintained in previous volumes. It contains concise and lucid accounts of recent medical and scientific work, particularly in therapeutics.

The ophthalmological section is ably dealt with by Sir Stewart Duke-Elder. Sections are devoted to the effect of bacterial lysate in the treatment of staphylococcal conjunctivitis; to dinitrophenol cataract, and cataract caused by vitamin deficiency; uveoparotitis; sympathetic ophthalmitis; lupus erythematosus associated with choroiditis; a method of employing an adjustable suture in cases of recession; and the treatment of optic atrophy by malaria, fever therapy (using the Kettering hypertherm) and sympathectomy.

The production and illustrations are excellent.

Controlled Reading: A Correlation of Diagnostic Teaching, and Corrective Techniques. By EARL A. TAYLOR. Pp. XXXVIII +367. Chicago: The University of Chicago Press. Cambridge: Cambridge University Press. 1937. 16s. net.

This book, which has been beautifully printed with very numerous illustrations, presents a study of the mechanical side of reading constituted by the alternate movement and fixation of the eyes across the page. Many experimentalists have been struck by the accurate adjustment of these movement habits in normal adult reading, and have endeavoured to record the movements. The method of photographing a narrow beam of light reflected from the surface of the cornea has proved the most fruitful. The author gives a list of the methods which have been used, with photographs of the apparatus. Brief accounts with diagrams of the mode of operation of this apparatus would have been helpful in showing the reader how this method has gradually developed. The author's own camera, the Ophthalm-O-Graph, is a particularly neat form of portable binocular eye-movement camera. One is not quite satisfied, however, as to whether the subject's eyes may not be distracted by the recording beams which shine right into them.

The author has photographed the eye movements of large numbers of children and college students, and many of these photographs are reproduced in the text. He is concerned to show how frequently irregular eye movements and fixations, and regressive

movements, are found among the slower more inefficient readers. It appears that this irregularity is sometimes related to ocular defect, and can be improved by the use of correcting lenses. But it can also be improved by direct re-education in reading habits. The reader practises reading material the words of which are exposed successively in regularly spaced groups by means of the Metron-O-Scope, a piece of apparatus designed and constructed by the author. It is claimed that eye movements become more regular, the perceptual span is increased, and hence the rate of reading improved. Whether in fact the actual comprehension of the reading material is improved seems more doubtful. It is very possible that reading difficulties are in fact due to difficulty in actual comprehension of the words; thus they are not caused by irregularity of eye movement, but are responsible for producing it. Nor should complete regularity of eye movement always be taken as a sign that adequate comprehension is taking place. It is possible for the eyes to move backwards and forwards regularly and mechanically across the printed page without the reader comprehending what he is reading; whereas rapid skimming and comprehension of the material may be accompanied by eye movements of extreme irregularity.

Thus though the author's treatment of the subject is most interesting and suggestive from the ophthalmological point of view, the psychological approach to the subject should not be forgotten.

CORRESPONDENCE

THE TEACHING OF ORTHOPTICS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In reference to a recent letter appearing in this Journal, the Orthoptic Board decided at their last meeting that students must, before commencing study, have passed the School Certificate, the Senior Certificate of the College of Preceptors, or have matriculated at a recognised University within the British Empire.

Yours very truly,

C. L. GIMBLETT.

May 31, 1937.

OBITUARY

S. A. KINNIER WILSON

THE death of Samuel Alexander Kinnier Wilson on May 12 at the early age of 58 years is a severe blow to neurology not only in Great Britain but also all over the world, and is a great loss to ophthalmology also.

Kinnier Wilson was born in the United States in 1878 and was educated in Edinburgh. He qualified M.B.Edin. in 1902 and served as house physician in the Edinburgh Royal Infirmary. In the following year he obtained the B.Sc. with honours in physiology. A research scholarship enabled him to put in post-graduate work at Paris; and on his return to England he became house physician at the National Hospital, Queen Square. At the expiration of this appointment he became resident medical officer, and later registrar and pathologist. He was elected to the honorary staff in 1913, became physician to out-patients in 1921 and physician to in-patients in 1925. It was not until 1912 that he took his M.D.Edin., securing the gold medal. His connection with the Royal College of Physicians began with his taking the M.R.C.P. in 1907; seven years later he became F.R.C.P., and in 1925 he gave the Croonian lectures. In 1912 he joined the staff of the Westminster Hospital and was at one time Dean of the Medical School. In 1919 he resigned his appointment at the Westminster Hospital on being elected junior neurologist at King's College Hospital.

It was in 1912 that he contributed to *Brain* the description of progressive lenticular degeneration which has ever since borne the name of Wilson's disease.

In 1920 he was appointed the first Editor of the *Journal of Neurology and Psychopathology*, a post which he held until the end of his life.

Wilson was the author of numerous papers on such neurological subjects as aphasia, epilepsy and narcolepsy. His purely ophthalmological writings were infrequent and were mainly concerned with the ophthalmoplegias. In 1921 at a combined meeting of the Neurological and Ophthalmological sections of the Royal Society of Medicine, Kinnier Wilson contributed to a discussion on "Ocular palsies"; he confined his remarks to the question of a possible unilateral cranial polyneuritis. In the same year he read a paper at the Annual Congress of the Ophthalmological Society of the United Kingdom on "Psychological peculiarities in certain visual auras in epilepsy." He had been a member of the Society since 1911. He was elected one of the secretaries in 1915 and served for the customary period of three years, when he became a member of the

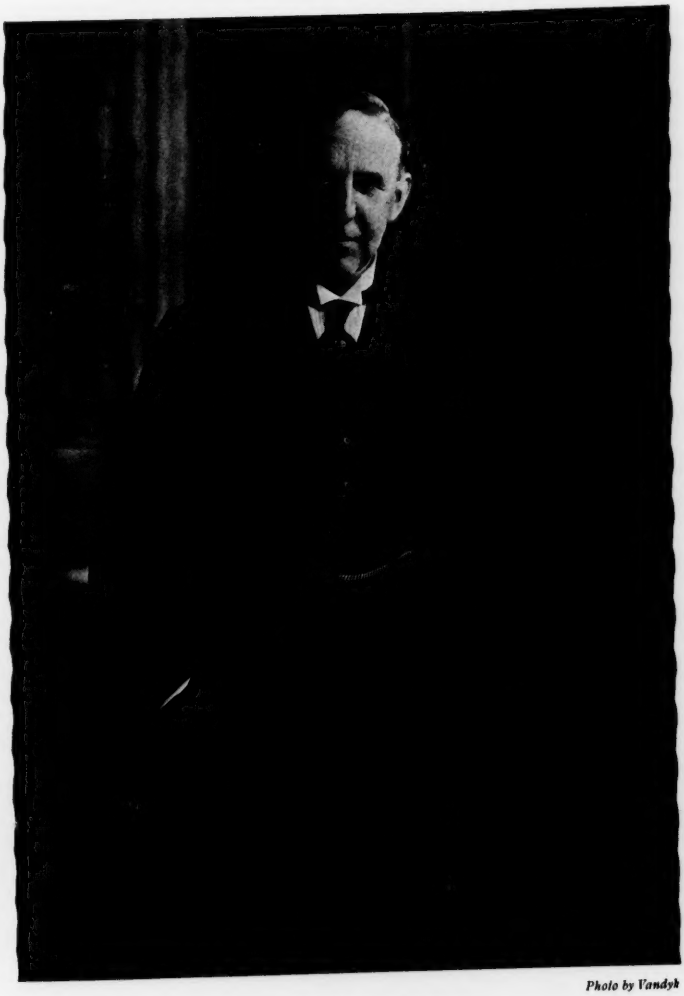

Photo by Vandyk

S. A. KINNIER WILSON

UNIV. OF MICH.

Council, and was succeeded as medical secretary by the present writer. Kinnier Wilson also served for many years as a trustee both of the Society and of the Nettleship Prize Fund.

In 1930 he gave the Morison lecture before the Royal College of Physicians at Edinburgh. Kinnier Wilson was a fluent speaker in French and German; he had a great reputation abroad as well as at home and was made an honorary member of many foreign Neurological Societies. As a lecturer, his power of lucid exposition of the most intricate problems of neurology has seldom been equalled. It was a pleasure to listen to him and when he was lecturing at Queen Square, the theatre was filled to capacity.

Our illustration, kindly lent by Mr. Paton, shows Kinnier Wilson as he was a few years back. In Stephen Paget's Life of Sir Victor Horsley is a photograph of the operating theatre at Queen Square in 1906, which shows amongst others, Kocher, of Berne, Horsley, S. A. K. Wilson, and "Loo" Powell, giving an anaesthetic with the Vernon-Harcourt inhaler.

NOTES

XV Concilium Ophthalmologicum Egypt, December, 1937

THE XVth International Congress is arranged for Cairo December 8—14, 1937. The two official subjects of the Congress will be:—

1. "Arterial hypertension of the retina." Introducers, H. Wagener and Keith. Reporters, Bailliart (clinical and physiological aspects), Koyanagi (pathological and anatomical aspects).
2. "Endocrinology and the eye." General introducer, Snapper. Reporters, von Szily, von Imre, Jeandelize, Lacarrère, Lo Cascio.

Two exhibitions are being arranged:—

1. A scientific exhibition, comprising anatomical and pathological specimens, slides, photographs, radiographs, etc.
2. A commercial exhibition, comprising instruments, apparatus, drugs, etc.

The Secretary General of the Congress is Dr. M. Tewfic. The fee for membership for a medical man is 50 Swiss francs. Fees for ladies and other associate members are 25 Swiss francs per person. The latter can attend receptions, excursions, etc., but cannot attend the scientific meetings, nor will they receive a copy of the Reports.

In connexion with the Congress the following information sent by Dr. Tewfic to Mr. MacCallan is of importance:—

"I have received, with pleasure, your letter dated April 19 and am glad to tell you that we have obtained 50 per cent. reduction

on the Egyptian State Railways for *members* and two of their accompaniers on condition that they must be wife, son or daughter.

We have as well a reduction of 20 per cent. on Syrian Railways.

We are in communication with Foreign Countries for reduction on their railways and with the Navigation Companies for reduction on their steamships."

N.B.—It is probable that for the word Syrian is to be understood Palestinian.

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Local Removal of Sarcoma of Ciliary Body

AT a recent meeting of the Vienna Ophthalmological Society as reported in the *Wiener Klin. Wochenschr.*, May 14, 1937, Lindner showed a case in which he had removed a sarcoma of the ciliary body from the eye. The procedure adopted was as follows:—Adhesions between the retina and choroid were produced by diathermy cauterizations over a circle extending far beyond the area of the growth. Fourteen days later a flap of sclera was turned back and the tumour was isolated and removed by means of the high frequency needle. At the same time the lens which had been damaged by the tumour was removed by means of a vectis. The eye recovered satisfactorily. Microscopically it was shown that the incisions had been carried out in healthy tissues. The tumour measured 4 by 8 mm.

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Corrigenda

Dr. I. C. MICHAELSON writes: on page 232, line 10 should read "negative duction at distance" instead of "negative duction at reading distance," and on page 233, the sixth line from the end "but many exophorias with low negative duction" should read "but many exophorias with low *positive* duction."

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Revue Internationale du Trachome and the International Organization against Trachoma

WITH the January issue (Vol. XIV, No. 1) the scope of *Revue Internationale du Trachome* has been enlarged. It is now the official organ of *La Ligue contre le Trachome* and of the International Organization against Trachoma. Mr. A. F. MacCallan is at present President of the International Organization against Trachoma and Dr. Wibaut, of Holland, is the Secretary. Membership of the Organization including postage of *La Revue Internationale du Trachome* is obtained by a subscription of 25 shillings sterling. The membership without the publication is 13 shillings sterling. Subscriptions should be paid to the account of the International Organization against Trachoma at the National Provincial Bank, 23, Wigmore Street, London, W.1. It will be recalled that the

Revue Internationale du Trachome is a quarterly publication of 64 pages per number. Articles are published in English, French and German. It includes an abstract section, one on reviews of books, meetings of societies and other notes. The editor is Dr. Paul J. Petit, 19, Rue de Bourgoyne, Paris.

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The Leslie Dana Gold Medal

THE medal, which is given annually for outstanding achievements in the prevention of blindness and the conservation of vision, has been awarded to Mrs. Winifred Hathaway.

FUTURE ARRANGEMENTS

1937

July 8-10.—Oxford Ophthalmological Congress, at Oxford.

October 1.—Midland Ophthalmological Society, at the Eye Hospital, Birmingham (Annual Meeting).

December 7.—Midland Ophthalmological Society, at Birmingham Eye Hospital (Middlemore Lecture).

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *May, 1937.*

GRIFFITH, JEFFERS, FEWELL and FRY. A study of the communication and direction of flow between cerebrospinal fluid and optic discs in the rat.

WRIGHT. Lectures on glaucoma. 1. Certain aspects of glaucoma.

LUCKIESH and MOSS. The avoidance of dynamic accommodation through the use of a brightness-contrast threshold.

BIELSCHOWSKY. The aetiology of squint.

LAMB. Chronic tuberculous uveitis.

WIENER and ALVIS. The use of concentrated-epinephrin preparations in glaucoma, iritis, and related conditions.

BARKAN and MAISLER. Adrenalin chloride 1:100 in ophthalmology.

KRAUSE. The spermine bases of ocular tissues.

FOLK. Paracentesis and atropine in the treatment of optic and retinal atrophies.

Annales d'Oculistique. *May, 1937.*

DESVIGNES. Syndrome of direct compression of the intra-cranial optic nerve.

MAMOLI. A case of retinal detachment with macular hole. Treatment by transbulbar diathermy.

DUPUY-DUTEMPS. Margino-cutaneous blepharopexy in lid repairs.

Zeitschrift für Augenheilkunde. *April, 1937.*

ADDA. A case of metastatic carcinoma of the ciliary body.
BÜCKLERS. Filiform cataract.
MAUKSCH. Pollen allergy as the cause of superficial keratitis.
BOKERMANN. Cure of glioma retinae through Röentgen rays.

Klinische Monatsblätter für Augenheilkunde. *April, 1937.*

LINKSZ. Dermotherapy in the daily practice of oculists.
BUSCHKE. Metastatic reticulo-endothelial sarcoma of the eyes.
POOS. A picture of small lymph-follicles in cavernous haemangioma of the orbit.
KURZ. Symmetrical iris tumour.
HORNIKER. Remarks on Dr. Sakae Kitahara's work, "clinical observations on the frequent occurrence of chorioretinitis centralis serosa in Japan."
LISCH. Rare forms of tapeto-retinal degeneration.
STREIFF. Effect of syntropans in normal and glaucomatous eyes.
SCHMELZER. Erosion of the eyes through concentrated tear gas.
WITTER. Congenital retinal atrophy as the suspected cause of axial hyperopia.
KISS. Visual acuity after bilateral cataract operation in early childhood.

Acta Ophthalmologia.

SCHUMACHER. The suppression of the monocular and binocular stimulus threshold during dark adaptation in the daytime and twilight apparatus.
LUNDBERG. A couple of cases of allergic conjunctivitis in urticaria patients.
FISCHER. Collected cases of herpes corneae in men.
NORDLÖW. Stability of cocaine solution during sterilization.
KRAVKOV. Effect of indirect light stimulation as a function of the intensity of a direct stimulus.
WITH. Is uveoparotitis a manifestation of benign lymphogranulomatosis?
VAN HEUVEN. The visual acuity in excessive illumination.
SKYDSGAARD. Bilateral tuberculous dacryoadenitis.

Bollettino d'Oculistica. *March, 1937.*

FEDERICI. Pathogenesis of post-operative delirium.
VITO. Experimental cataract of alimentary origin.
RUSSO. Optic neuritis in measles.
FABOZZI. Neuro-hypophyseal infantilism and hard capsulo-lenticular cataract.
SPINELLI. Pathogenesis of retinal tears in the genesis of detachment. (Deductions from ophthalmoscopic observations of the formation of holes).
BARATTA. Congenital familial changes in the iris.

Revista Oto-Neuro-Oftalmologica. *March-April, 1937.*

FASIANI and BELLONI. Surgery of the intracranial course of the optic canal

Anales de la Sociedad Mexicana de Oftalmologia y Oto-Rino-Laringologia.

January-March, 1937.

BAUER. Heine's cyclodialysis in the treatment of glaucoma.
IRYS. Clinical history of a case of Leishmaniasis of the eyelid.
ESPINO. Hereditary familial amaurotic idiocy in Mexico.
ESTRADA. Some rules for the treatment of pterygium.